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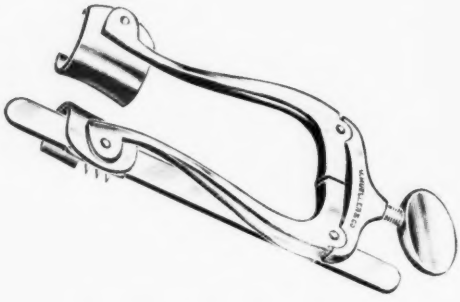

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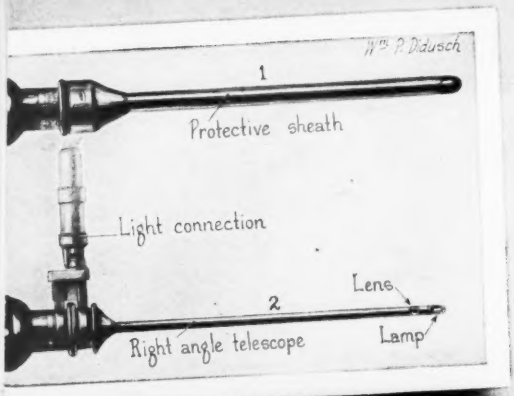

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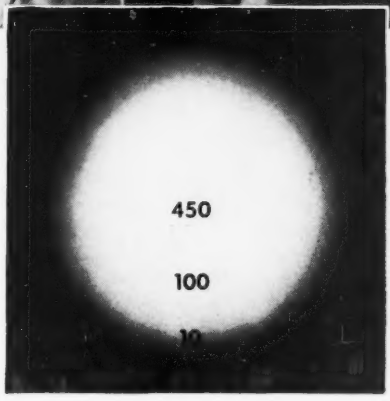

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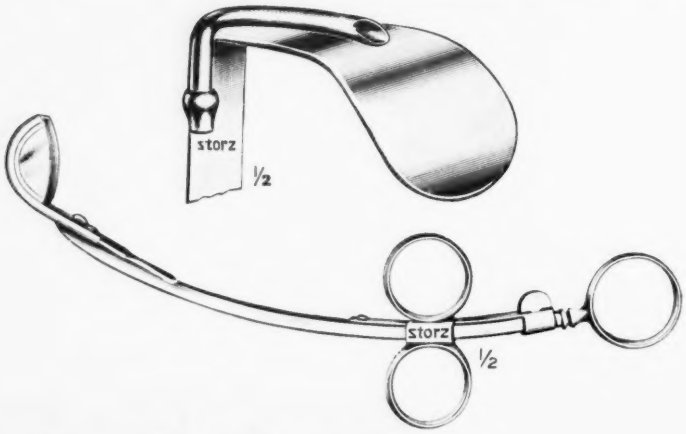

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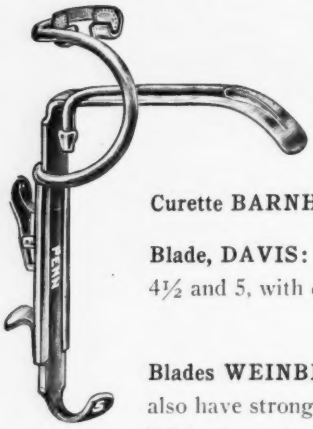

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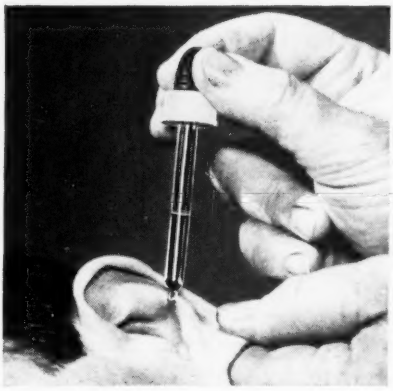

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*Laryngoscope 58:1279 (Dec.) 1948.

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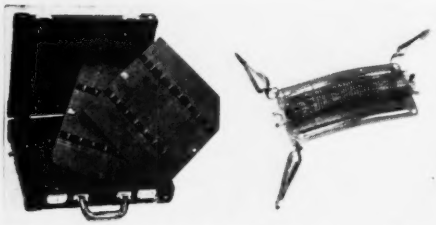

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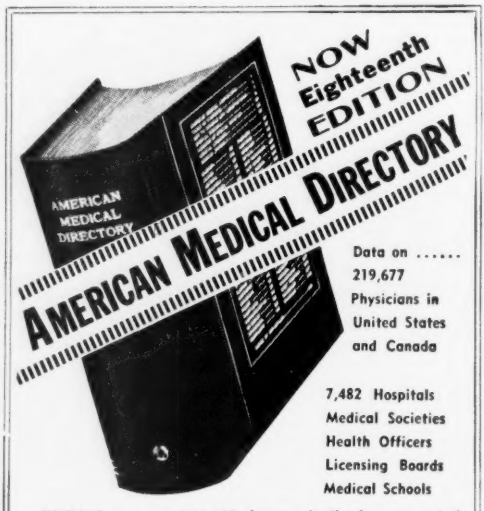

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VOLUME 58 NOVEMBER 1953 NUMBER 5

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SELECTED PROBLEMS IN THE DIAGNOSIS OF LARYNGEAL CARCINOMA

STANTON A. FRIEDBERG, M.D.
AND
LINDEN J. WALLNER, M.D.
CHICAGO

THIS report is the direct outgrowth of efforts to photograph various types of laryngeal pathology for teaching purposes. In the course of accumulating and reviewing pictures we were impressed by the problem of leucoplakia in its varied forms and associated incidence of carcinoma of the vocal cord. The relative lack of symptoms in the extracordal or extrinsic cancer and the amazing extent to which such lesions had often progressed before recognition were consistently disturbing observations. We became interested in correlating mirror and direct laryngeal views with operative specimens of the same patient. The striking similarity between the gross appearance of some forms of tuberculosis of the larynx and carcinomas of the larynx was evident on numerous occasions. Inasmuch as the choice of therapy often involved problems in diagnosis, some consideration of treatment has been included. For a more definitive analysis of these particular topics than was afforded by photography, we have gathered from our records such statistics and clinical data as may seem pertinent.

MATERIAL AND CLASSIFICATION

The study is based on a series of 116 patients with carcinoma of the larynx observed in the past five years.

We have followed, with minimal modification, the classification proposed by Walsh.[1] To avoid varied interpretations of the term "intrinsic carcinoma," however, we prefer the designation "cordal carcinoma" for tumors confined to either one or both true vocal cords, with normal cord mobility.

When a tumor involves the true cord, the ventricle or the ventricular band or extends just inferiorly from an initial cordal origin, we favor the term "endolaryngeal" to distinguish it from cordal carcinoma. In most endolaryngeal carcinomas the true cord is fixed or its motion is impaired, although this is not uniformly true.

A distinct and separate entity is the type of growth which arises primarily from the subglottic space. Such tumors may extend superiorly to involve the true vocal cord or inferiorly to invade the upper trachea.

Finally, the terms extracordal and extrinsic are used synonymously to denote a primary lesion of the epiglottis, arytenoid, aryepiglottic fold or pyriform sinus. This

Presented at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 15, 1952.

From the Presbyterian Hospital; the City of Chicago Municipal Tuberculosis Sanitarium; the Department of Otolaryngology, University of Illinois College of Medicine, and the Veterans Administration Hospital, Hines, Ill.

1. Walsh, T. E.: The Classification of Carcinoma of the Larynx, Laryngoscope **57**:414-418 (June) 1947.

group comprises the largest category of malignant growths of the larynx in our series and poses the greatest difficulty in respect to early recognition and adequate therapy.

Laryngeal neoplasms are sometimes so extensive that their site of origin cannot be ascertained. Under such circumstances, a careful history may aid in determining the original location.

In the entire series there were 113 male patients and 3 female. (The incidence of carcinoma of the larynx in females varies from 3 to 10%. Our series includes a

TABLE 1.—*Analysis of Laryngeal Carcinoma in 116 Patients*

Types of Carcinoma	Patients	
	No.	%
Cordal (occurs in true cords, with normal mobility)	31	26.7%
Endolaryngeal (occurs in a true cord, ventricle, false cord or early subglottic invasion)	18	15.5%
Subglottic	9	7.7%
Extracordal or extrinsic (occurs in the epiglottis, arytenoid, aryepiglottic fold or pyriform sinus)	58	50.0%

number of male patients observed in a Veterans' Hospital.) The average age for the group was 56.7 years, although this figure was somewhat higher for patients with subglottic carcinoma.

It will be observed from Table 1 that the ratio between intralaryngeal and extrinsic lesions corresponds to the range of distribution reported by other authors.[2]

2. (*a*) McCall, J. W., and Fisher, W. R.: Carcinoma of the Larynx: A Report of 194 Cases with 149 Laryngectomies, Laryngoscope **62**:475-485 (May) 1952. (*b*) Martin, H.: Cancer of the Larynx, in Surgery of the Nose and Throat, edited by J. D. Kernan, New York, Thos. Nelson & Sons, 1942, pp. 431-505.

EXPLANATION OF FIGURES 1 TO 12

Fig. 1.—Keratosis of the larynx involving anterior half of left true cord. There was no recurrence three years after removal.

Fig. 2.—Extensive hypergeratotic papilloma of left true cord.

Fig. 3.—Larynx of a patient who had smoked excessively for 54 years. Bilateral leucoplakia, with subglottic mass at anterior commissure. Biopsy revealed carcinoma.

Fig. 4.—Leucoplakia and infiltration of anterior portion of right true cord in a heavy smoker. (Right cord stripped for leucoplakia six years previously.) Biopsy revealed carcinoma.

Fig. 5.—Leucoplakia and infiltration of right true cord of an excessive smoker with hoarseness for three months. Biopsy revealed a carcinoma arising from leucoplakia.

Fig. 6.—Cordal carcinoma, with lesion limited to anterior half of left true cord. Mobility was normal. There was no recurrence after thyrotomy in which the involved side, anterior commissure, and part of the opposite cord were excised.

Fig. 7.—Lesion of the epiglottis of a patient with pulmonary tuberculosis, discovered when the patient requested removal of wax from his ears. Biopsy revealed carcinoma.

Fig. 8.—Proliferative carcinoma on laryngeal surface of epiglottis in a patient with diabetes, gangrene, and severe cardiac disease.

Fig. 9.—Postirradiation view of previous larynx. All evidence of tumor had disappeared. There has been no recurrence for three years.

Fig. 10.—Extensive carcinoma of left pyriform sinus in a patient with a painless cervical node and no other symptoms.

Fig. 11.—Infiltration and ulceration of right arytenoid in a patient with symptoms of two weeks' duration. Biopsy revealed a squamous-cell carcinoma.

Fig. 12.—Infiltration and ulceration of left arytenoid and aryepiglottic fold in a patient with pulmonary tuberculosis. Lesion completely disappeared after streptomycin therapy.

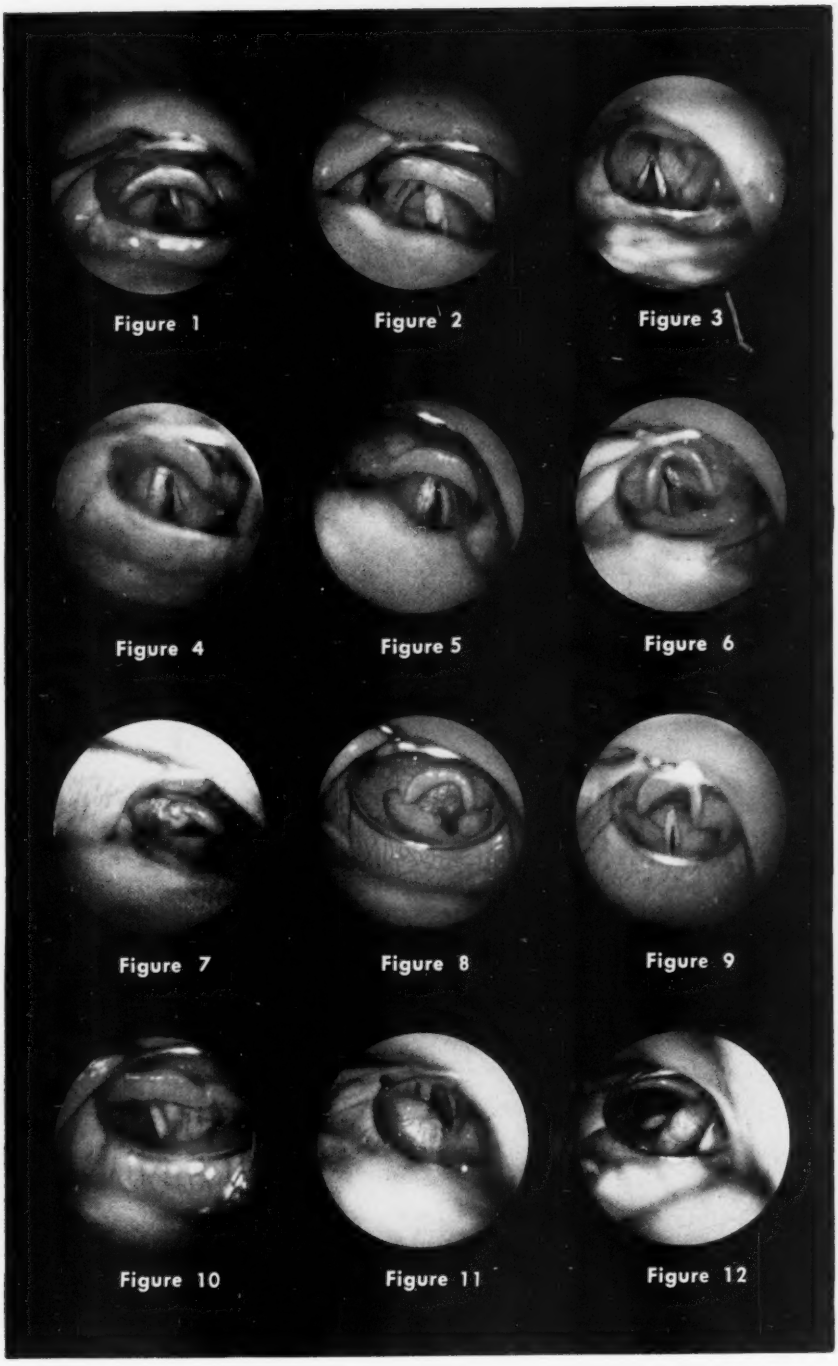

Figure 1

Figure 2

Figure 3

Figure 4

Figure 5

Figure 6

Figure 7

Figure 8

Figure 9

Figure 10

Figure 11

Figure 12

CORDAL (VOCAL CORD) CARCINOMA

As previously stated, the category of cordal carcinoma embraces only those lesions which are limited to the true cord (Figs. 4, 5, 6, and 13), may or may not traverse the anterior commissure, and do not interfere with normal mobility. There were 31 patients in this group, including the only 3 females represented in the entire series. The average age of the group was 56.2 years. Hoarseness was the only symptom and was present in every patient.

Each member of the group except one gave a history of smoking, and two-thirds of these could be regarded as heavy smokers (in excess of one package of cigarettes daily). The incidence of smokers is thus considerabl higher than the 73.7% figure quoted by Wynder and Graham [3] as representing the smokers among a general (noncancerous) hospital population.

The duration of the symptom of hoarseness before diagnosis was an average of seven months. (This figure does not include 20% of the group who had been hoarse for years.) Six of the 31 patients had been treated with antibiotics, sprays, gargles, lozenges, and the like during the period in which it could be presumed that evidence of a malignant growth might well have been at hand. The factors of patient neglect and shortcomings of the medical profession, as outlined by Pack [4] and Robbins,[5] are apparent in these statistics. Ferguson [6] has recently suggested that the widespread use of antibiotics may be responsible for a definite trend away from early diagnosis in laryngeal cancer. The delay in diagnosis in this series emphasizes Ferguson's contention.

One of the patients in this group had a second primary carcinoma. None had palpable cervical lymph nodes.

Leucoplakia and Cordal Carcinomas (Figs. 3, 4, 5, 13, and 14*A* and *B*).—Among the 31 patients with cordal carcinoma there were 19 (61%) who revealed definite evidence of leucoplakia, and in 9 of the 19 the condition was bilateral. The majority of these patients were excessive smokers (two packages of cigarettes or more per day). LeJeune [7] has noted that leucoplakia is being observed more frequently than in former years and that all of his patients with leucoplakia of the vocal cords were excessive users of tobacco. Similar observations on the relationship between leucoplakia and the excessive use of tobacco have been made by Clerf, Putney, and O'Keefe.[8]

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3. Wynder, E. L., and Graham, E. A.: Tobacco Smoking as a Possible Etiologic Factor in Bronchiogenic Carcinoma: A Study of 684 Proved Cases, J. A. M. A. **143**:329-336 (May 27) 1950.

4. Pack, G. T., and Gallo, J. S.: Culpability for Delay in the Treatment of Cancer, Am. J. Cancer **33**:443-462 (July) 1938.

5. Robbins, G. F.; Conte, A. J.; Leach, J. E., and MacDonald, M.: Delay in Diagnosis and Treatment of Cancer, J. A. M. A. **143**:346-348 (May 27) 1950.

6. Ferguson, G. B.: Recent Trends in the Diagnosis and Treatment of Cancer of the Larynx, North Carolina M. J. **12**:23-25 (Jan.) 1951.

7. LeJeune, F. E.: Surgical Treatment of Early Carcinoma of the Larynx, Tr. Am. Laryng. Rhin. & Otol. Soc. **55**:318-325, 1951.

8. Clerf, L. H.; Putney, F. J., and O'Keefe, J. J.: Carcinoma of the Larynx, Laryngoscope **58**:632-641 (July) 1948.

Unless marked infiltration of the vocal cord has occurred, it is often difficult to evaluate the status of a leucoplakic site. This was ably described by Negus [9] in 1939:

> Localized or diffuse overproduction of the superficial epithelium offers considerable difficulty in deciding whether malignant changes are commencing in the deeper layers of what is a potentially precancerous condition.

As an illustration, the following case may be cited:

> T. W., a 59-year-old man, had been hoarse for five months. There was leucoplakia of the free edge of the middle third of the left vocal cord (Fig. 13*A*). Three biopsies were reported as showing only epithelial hyperplasia. Because of the recurrence of leucoplakia anterior to the original site (Fig. 13*B*), a thyrotomy was performed, and the vocal cord was removed. Serial sections disclosed epithelial hyperplasia in addition to carcinoma in situ but no subepithelial invasion (Fig. 13*C*). The patient died in an accident two years later, without clinical evidence of recurrence.

It is our feeling that an aggressive plan of treatment should be undertaken in a case in which there exists any reasonable doubt as to the possibility of cancer.

The role of chronic irritation in precancerous conditions of the larynx has been emphasized repeatedly by Jackson and Jackson.[10] Clerf [11] refers to keratosis, hyperkeratosis, or leucoplakia, as primarily a localized benign hyperplasia of the epithelium which does not invade the submucosa. In discussing etiology he points to the predominance of the condition in males and to the immoderate use of tobacco (although there is no definite proof that this is a causative factor). Alcohol is of doubtful influence. Most of the patients used their voices excessively or abnormally. Chronic laryngitis is a well-recognized predisposing factor, and this has been substantiated by New and Erich's [12] histologic demonstration of the marked subepithelial inflammatory reaction observed in these conditions. Dietary deficiency has been thought to be of significance in some patients.[13]

While leucoplakia and keratosis are usually considered in discussions of benign growths in the larynx,[14] it is equally true that references to them are found in almost all dissertations on laryngeal carcinomas. Their intimate relation to cancer and the necessity for frequent examination of the larynx in these patients cannot be overemphasized.[15] it is well known that simple keratoses may be removed and not recur, that they may reappear in benign form after removal in some patients, and that the changes of malignancy may supervene slowly or rapidly in others. There is no way of predicting the sequence of events in any given situation.

> W. C., a 54-year-old man and an excessive smoker, had been hoarse for nine months. During most of this period he had received antibiotics as treatment for this difficulty. A whitish pro-

9. Negus, V. E.: Differential Diagnosis of Intrinsic Carcinoma of the Larynx, Arch. Otolaryng. **29**:223-234 (Feb.) 1939.

10. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937, pp. 370-377.

11. Clerf, L. H.: Keratosis of the Larynx, J. A. M. A. **132**:823-826 (Dec. 7) 1946.

12. New, G. B., and Erich, J. B.: Benign Tumors of the Larynx: A Study of 722 Cases, Arch. Otolaryng. **28**:841-910 (Dec.) 1938.

13. Graham, H. B.: Keratosis of the Larynx, Arch. Otolaryng. **36**:735-739 (Nov.) 1942.

14. Holinger, P. H., and Johnston, K. C.: Benign Tumors of the Larynx, An. Otol. Rhin. & Laryng. **60**:496-509 (June) 1951. New and Erich.[12]

15. Gordon, G. R.: Keratosis of the Larynx: Report of Case with Underlying Carcinoma in situ, Laryngoscope **60**:1201-1209 (Dec.) 1950.

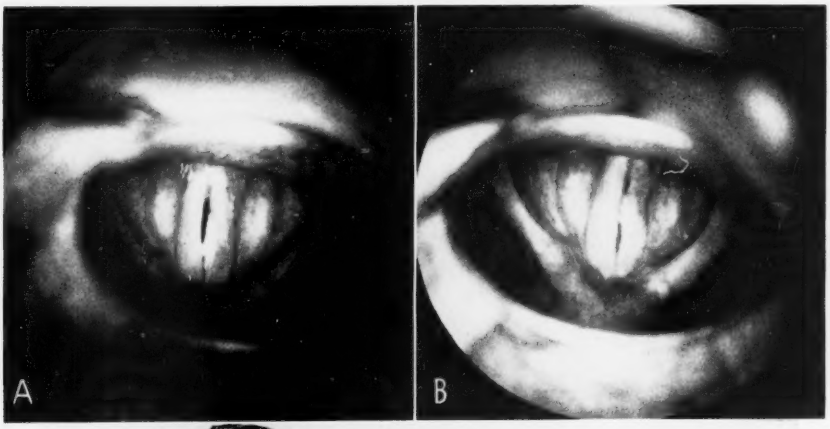

Fig. 13.—*A*, leucoplakia involving middle third of left true vocal cord; biopsy revealed epithelial hyperplasia; *B*, recurrence of leucoplakia in same patient anterior to the original site of removal; *C*, section from same patient through true vocal cord removed by thyrotomy; hyperkeratinization, epithelial hyperplasia, and carcinoma in situ (hematoxylin and eosin stain; × 90).

liferative area was noted on the anterior half of the left true vocal cord (Fig. 1). This was removed and showed epithelial hyperplasia, hyperkeratinization, and a submucosal inflammatory reaction. No recurrence has been observed in over three years.

The rapid development of a malignant growth upon a leucoplakic anlage is illustrated by the following case:

L. Z., a 55-year-old man with a history of heavy smoking for years, had been hoarse for only three months. On laryngeal examination there was observed a white granular elevation

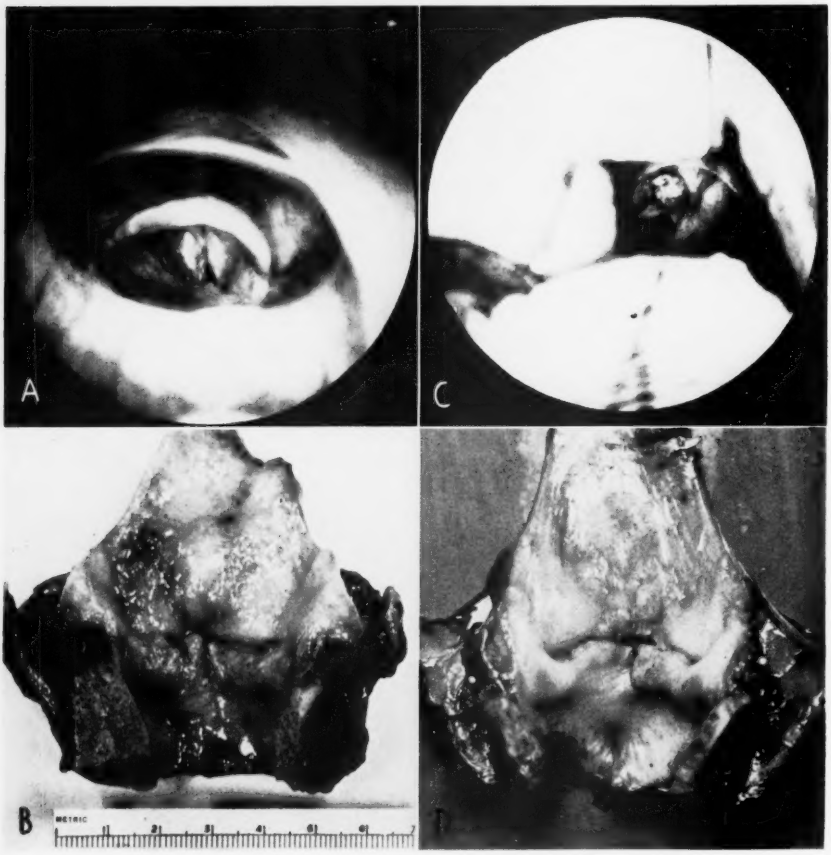

Fig. 14.—*A*, pachydermia of the larynx with whitish pebbly irregularity of both vocal cords and normal mobility; fourth biopsy disclosed carcinoma; *B*, specimen from same patient taken at laryngectomy; bilateral carcinomatosis of true vocal cords and interarytenoid space; *C*, a view by means of suspension laryngoscopy of proliferative lesion involving right vocal cord (impaired motion of cord was evident clinically); *D*, operative specimen from same patient, revealing extension of lesion to ventricle, ventricular band, base of epiglottis, and across anterior commissure: endolaryngeal carcinoma.

along the superior surface of a markedly thickened right true vocal cord (Fig. 5). Motion of the cord was not impaired. There were also leucoplakic alterations on the opposite cord. Biopsy of the right cord disclosed a squamous-cell carcinoma arising from an extensive leucoplakic infiltration.

In contrast, malignant transformation may occur after a long interval:

C. H., a 44-year-old salesman and a heavy smoker, gave a history of having been hoarse for eight years. He had undergone a stripping operation on his right vocal cord six years previously and had been informed that the tissue had shown leucoplakia. When he was observed in 1949, there was a whitish mass involving the right true cord, extending posteriorly to the vocal process of the arytenoid and anteriorly to the commissure, at which point it projected slightly (Fig. 4). Some subglottic thickening was evident, but mobility of the cord was not impaired. Biopsy showed a squamous-cell carcinoma. The entire larynx was reddened, and there was leucoplakia of the opposite cord. There were no palpable cervical nodes. Laryngectomy was performed in 1949. It was found that the lesion extended just across the anterior commissure, invaded the floor of the ventricle, and reached the arytenoid posteriorly. The patient has remained well since surgery.

Four patients with similar histories were carefully followed by Clerf [11] for two to four years, three eventually requiring partial laryngectomy and one a complete laryngectomy.

Pachydermia of the larnyx represents another form of diffuse epithelial hyperplasia with potential malignant alteration. This condition usually appears in the posterior commissure but may later become evident along the entire glottic rim.[16]

C. S., a 58-year-old man, had been intermittently hoarse for one year and persistently so for six months prior to admission. There was a whitish, pebbly irregularity involving both true vocal cords and the interarytenoid space, with normal cord mobility (Fig. 14*A*). A diagnosis of carcinoma was made on the fourth biopsy, the previous ones having shown only epithelial hyperplasia. The specimen taken after the laryngectomy revealed diffuse carcinomatosis limited to the true vocal cords and interarytenoid space (Fig. 14*B*). The patient has remained well for two and one-half years after surgery.

The prognosis of cancer of the true vocal cord is excellent, probably because of the early appearance of a change in voice and the relatively scant lymphatic drainage.

Endolaryngeal removal of certain small localized lesions, irradiation, and partial or total laryngectomy all have their proponents and specific indications, but the careful selection of cases is the principal assurance of success in dealing with cordal lesions. The already high rate of cure of cordal cancer will probably be improved if, through education, we can shorten the average seven-month interval between the onset of hoarseness and the inception of definitive care, if adequate examination is substituted for inapplicable therapeutic measures, and if laryngologists insist on the guarded scrutiny of patients with leucoplakia, keratosis, and pachydermia—the so-called precancerous conditions involving the larynx.

ENDOLARYNGEAL CARCINOMA

Endolaryngeal carcinomas are those tumors which involve the true vocal cord, the ventricle, the ventricular band or the immediate infraglottic area (Figs. 4, 14*C* and *D*, and 15*A* and *B*). Impaired mobility or fixation of the true cord is often present, though not invariably so. There were 18 such patients with an average age of 55.2 years. All were males; the symptom of hoarseness was present in each, but in contrast to cordal carcinoma, six patients, or 33%, also mentioned the sensation of throat discomfort. Smoking was recorded in the history of all but four, for whom this information was not available. The average duration of symptoms before diagnosis was 8.7 months. Only two patients had been treated irrelevantly before admis-

16. Grossman, A. A., and Mathews, W. H.: Keratosis of the Larynx with Progression to Malignancy, Canad. M. A. J. **66**:39-41 (Jan.) 1952.

sion. Leucoplakic alterations were evident in the larynges of four patients, all heavy smokers. One patient had pulmonary tuberculosis in addition to carcinoma of the larynx. None of the patients had palpable cervical nodes, nor were there any in whom a second primary malignant lesion was evident.

Whereas it is not difficult to judge fairly accurately the extent of a cordal carcinoma, the degree of involvement of an endolaryngeal tumor is more complex and

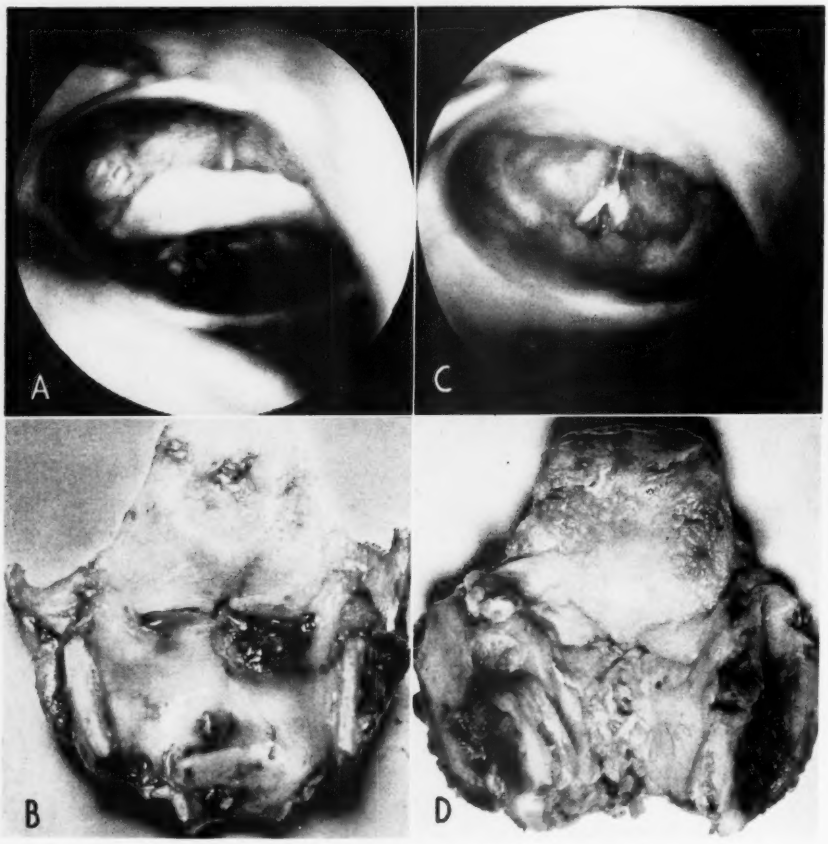

Fig. 15.—*A*, mirror view of ulcerating carcinoma of right hemilarynx with subglottic extension; *B*, operative specimen from same patient; note correlation with preoperative estimate of endolaryngeal lesion; *C*, mirror view of subglottic mass obstructing airway in patient who had been under treatment for bronchial asthma; *D*, operative specimen from same patient, revealing extensive subglottic carcinoma; additional tracheal rings were removed (not shown on specimen).

becomes a matter of extreme importance in respect to therapy. Thus we endeavored to correlate the laryngeal appearance with photographs of operative specimens in an effort to establish some familiarity with this type of evaluation.

H. F., a 70-year-old man who had been an excessive smoker for 50 years, had noticed hoarseness for six weeks. Mirror, direct, and suspension laryngoscopy disclosed a proliferative

lesion of the right true vocal cord throughout its entire length, with impaired mobility but no palpable cervical nodes (Fig. 14*C*). The operative specimen after laryngectomy revealed extension of the tumor to the ventricle, ventricular band, base of the epiglottis, and across the anterior commissure (Fig. 14*D*). It was evident that the tumor was more extensive than had been suspected clinically.

Another example of this attempted correlation is illustrated by the following case:

J. M., a 54-year-old man, had been a heavy smoker for 40 years. He had been hoarse for two years. Laryngeal examination revealed an ulcerative lesion of the right true vocal cord with subglottic extension (Fig. 15*A*). The vocal cord was immobile. There were no palpable cervical nodes. The specimen taken after laryngectomy disclosed an extension of the tumor into the ventricle and subglottic space, correlating well with the preoperative clinical estimate (Fig. 15*B*).

It is our belief that repeated examination of any larynx in this category is often desirable and even necessary to properly assess the degree of pathological involvement prior to the choice of surgery. As diagnostic aids, suspension laryngoscopy and the use of other types of self-retaining laryngoscopes provide invaluable means of unhurried study. In addition to the usual x-rays, planigrams are particularly useful in outlining subglottic extension in these cases.

These added studies might conceivably result in fewer attempts to extend the indications for laryngofissure in a questionable situation, though we believe that such a procedure is rarely, if ever, indicated in endolaryngeal carcinoma. Of even greater importance, however, is the decisive choice between wide field laryngectomy and laryngectomy combined with radical neck dissection. The subsequent appearance of cervical metastases not clinically evident at the time of the original surgery seems most prevalent in cases of endolaryngeal carcinoma.[8] Recent studies [17] point to the desirability of routine combined laryngectomy and neck dissection for treatment of extensive endolaryngeal cancer, reserving wide field laryngectomy for cordal lesions which have involved the ventricular or subglottic areas to a limited degree.[17] It should also be added that conservative, or so-called narrow field, laryngectomy for treatment of extensive endolaryngeal carcinoma is fraught with considerable risk and is mentioned only to discourage its use in these cases.

SUBGLOTTIC CARCINOMA

Nine male patients comprised the group of subglottic carcinoma, the average age being 62.8 years. All had been hoarse, and four presented evidence of obstructive dyspnea, three of these requiring emergency tracheotomy on admission. There was a history of heavy smoking on the part of six patients; no information on this point was available on three. Of particular interest was the 13.4-month average duration of symptoms before diagnosis. During this interval two of the patients had been treated unsuccessfully for bronchial asthma. Leucoplakia was evident in two patients. One patient's laryngeal cancer was his second primary cancer.

Subglottic carcinoma (Figs. 3 and 15*C* and *D*) does not usually produce symptoms until the lesion has invaded the vocal cord or has caused some narrowing of the airway, as must be evident from the figures in the preceding paragraph. In

17. Ogura, J. H., and Bello, J. A.: Laryngectomy and Radical Neck Dissection for Carcinoma of the Larynx, Laryngoscope **62**:1-52 (Jan.) 1952.

addition to careful direct laryngoscopy (for these lesions may be easily overlooked on mirror examination), planigrams of the larynx are helpful in outlining sugglottic neoplasms.

J. B., a 65-year-old man, had been under treatment for bronchial asthma for nine months. He had been hoarse for seven months. He was known to have diabetes mellitus. Respiratory obstruction and deep cyanosis were evident upon his emergency admission from another hospital. A tracheotomy was performed after rapid visualization of a subglottic mass obstructing the airway (Fig. 15*C*). Wide-field laryngectomy was performed eight days later with removal of additional tracheal rings (not shown on specimen) (Fig. 15*D*). Postoperative irradiation was given. Although there has been no evidence of local or metastatic neoplasm in two years, we feel, in retrospect, that laryngectomy combined with neck dissection would have been the procedure of choice in this situation.

Because of the relatively rich lymphatic drainage from the infraglottic area and the frequent delay in diagnosis, subglottic carcinoma is best treated by laryngectomy or laryngectomy plus block neck dissection. (An indication of the changing concepts in this regard is to be found in the writings of St. Clair Thomson,[18] who reported six cures among 13 patients with subglottic carcinoma treated by laryngofissure.) Irradiation is regarded as ineffective against subglottic growths.

F. S., a 69-year-old man, had been hoarse for one year. He had been an excessive smoker since the age of 15 and had severe chronic bronchial asthma of an allergic type. There had been one and possibly two episodes of coronary thrombosis. The larynx was characterized by diffuse bilateral cordal leucoplakia and a small subglottic mass beneath the anterior commissure (Fig. 3). Irradiation was given because of his extremely poor general condition. There was a resulting chondronecrosis with severe localized pain and dysphagia for eight months before he died of inanition. Autopsy disclosed a large area of necrosis involving the larynx and esophagus. Microscopic studies revealed no tumor residue, either locally or in the cervical lymphatics.

Although the preceding synopsis is not intended to discredit the merits of irradiation therapy, it is apparent that the dose required for tumor sterilization also resulted in tissue necrosis, with prolonged suffering and the eventual death of the patient. This might have been avoided by surgery, even though the operative risk was great.

EXTRACORDAL (EXTRINSIC) CARCINOMA

In the category of extracordal carcinoma are to be found those patients with primary lesions of the epiglottis, arytenoid, aryepiglottic fold or pyriform sinus (Figs. 7, 8, 10, and 11). Various terms have been used to designate this group of laryngeal malignant growth, among which "extrinsic carcinoma" is probably the most familiar. Orton[19] has suggested that they be referred to as "epilaryngeal tumors." In advocating the expression "extracordal," however, we are thinking of a sharp distinction between these and cordal, endolaryngeal or subglottic growths, in order to obviate the varied interpretations hitherto accorded the terms "intrinsic" and "extrinsic." Perhaps an international otolaryngological organization will some day take up the problem of nomenclature of laryngeal tumors, as was done in the realm of segmental bronchial anatomy.

18. Thomson, St. C.: Subglottic Cancer of the Larynx, J. Laryng. & Otol. **52**:803-816 (Dec.) 1937.

19. Orton, H. B.: Treatment of Extensive Carcinoma of the Larynx, Tr. Am. Laryng. Rhin. & Otol. Soc. **55**:326-340, 1951.

Of the 58 patients represented in this group, all were males, and the average age was 56.3 years. The commonest symptom was some form of throat discomfort which was present in 40 patients (69%). Among this number, 18 complained of actual dysphagia, 7 of localized pain, and 2 of the sensation of a foreign body. Hoarseness was the next most frequent symptom, occurring in 35 patients (60%). There were 47 habitual smokers, 9 moderate smokers, and 2 nonsmokers in the group.

In this group of 58 patients with extracordal carcinoma, there were 5 who presented two distinct primary malignant tumors. It will be recalled that one patient with cordal cancer and another with subglottic cancer also had second primary lesions. Multiple primary carcinomas are by no means rare, and the 6% incidence in our 116 patients is comparable to the figures from autopsy reports on much larger series.[20] Warren and Gates [21] stated that "an individual with one cancer is more apt to develop a second cancer than would be expected by chance alone." This tendency was thought to be 11 times greater than the tendency of a patient without cancer to manifest his first lesion.

The average duration of symptoms before diagnosis was eight months. Eight patients (14%) had been treated by measures which indicated inadequate examination.

Considering the abundant lymphatic supply of the extracordal portions of the larynx and the paucity of symptoms due to the relatively silent areas of involvement, it is not surprising to find these lesions far more extensive than either cordal or endolaryngeal growths. Thus, five patients in the group had as their only complaint a painless swelling in the neck (subsequently proved to be metastatic carcinoma). In the entire group of 58 patients, 20 (34.5%) were found to have cervical metastases on admission, and in 3 the metastases were bilateral.

Even more striking in this connection was the discovery of extensive neoplasms in four patients undergoing routine hospital examinations for unrelated illnesses.

A 61-year-old man entered the hospital for hernia repair. A firm nodule was noted in both cervical regions. He was found to have a large ulcerating carcinoma of the epiglottis and cervical metastases.

Another patient, also hospitalized for hernia surgery, had unilateral cervical metastasis from a primary carcinoma of the epiglottis.

The third patient was in a tuberculosis sanitarium and requested removal of the wax from his ears. An ulceration of the epiglottis, presumably tuberculous, was found on biopsy to be a squamous-cell carcinoma (Fig. 7).

A fourth patient with painless cervical swelling was found to have an extensive carcinoma of the pyriform sinus and metastases (Fig. 10).

At the present time extracordal carcinoma has the poorest prognosis of any type of laryngeal cancer. There are many patients in whom the results of irradiation are rather remarkable, but it is impossible to predict the response in any given situation. Circumstances sometimes allow no choice:

N. K., a 52-year-old man with amyotrophic lateral sclerosis, was found to have an extensive carcinoma involving the laryngeal surface of the epiglottis and left false cord. He received

20. Judge, A. F.: Multiple Primary Malignant Tumors: Report of 3 Cases Encountered on Otolaryngological Service, Ann. Otol. Rhin. & Laryng. **59**:614-621 (Sept.) 1950.

21. Warren, S., and Gates, O.: Multiple Primary Malignant Tumors: A Survey of the Literature and a Statistical Study, Am. J. Cancer **16**:1358-1414 (Nov.) 1932.

irradiation therapy, resulting in the complete disappearance of the tumor. One year later he died of his primary illness. At autopsy there was neither gross nor microscopic evidence of any laryngeal tumor or cervical metastasis.

In another instance the patient's general condition was also a contraindication to surgery:

J. A., a 72-year-old diabetic with gangrene and marked arteriosclerotic heart disease, had noted hoarseness and painful swallowing for four months. There was an extensive carcinoma of the buccal mucosa and a second primary malignant growth involving the epiglottis (Fig. 8). Irradiation of the larynx produced complete regression of the tumor (Fig. 9). The oral lesion was similarly treated. There has been no recurrence of either for two years.

In contrast to these examples, the following case may be cited:

E. T., a 55-year-old man, noticed throat discomfort and hoarseness for two months. He was found to have an ulcerating carcinoma involving the epiglottis, left aryepiglottic fold, and pyriform sinus. Surgery was refused, and irradiation was substituted. There was an excellent response for five months, followed by reappearance of the tumor on the arytenoid. A combined laryngectomy and block dissection was performed. Esophageal fistula resulted but eventually healed, and the patient has been well for two years.

With full realization of the gravity of the prognosis in these cases, current efforts are being made to improve the rate of cure by means of surgery. Certain patients whose lesions were hitherto irradiated as a palliative measure (with hope but no assurance of a favorable response) are now regarded as likely candidates for en bloc resection of the cancer-bearing areas.

W. B., a 54-year-old man, had noticed hoarseness and throat discomfort for six months. Treatment with gargles and antibiotics had been given ineffectively for four months. On admission he was found to have an extensive carcinomatous ulceration involving the left ventricular band, aryepiglottic fold, and pyriform sinus. Laryngectomy and neck dissection were carried out in continuity. The patient has remained free of disease for two years.

More extensive procedures involving resection of the pharynx and esophagus have been recommended by Brunschwig,[22] Orton,[23] and others.

In summarizing current thought regarding treatment of extracordal carcinoma, it must be reemphasized that the prognosis is poor and will probably not improve appreciably until some form of cancer detection test insures earlier recognition. Although there is a definite place for irradiation therapy in cancer of the larynx, the most favorable results reported by radiologists have not compared with surgical figures.[24] (In all fairness it should be mentioned that surgical figures deal principally with operable cases, while radiologic analyses must include operable, inoperable, and recurrent cases.[25]) The patients deemed most suitable for surgery are also those most likely to obtain good results from irradiation.[26] And finally, the rate of salvage with improved surgical management is appreciably greater than that with the most optimistic irradiation results.

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22. Brunschwig, A.: Panlaryngectomy for Advanced Carcinoma of the Larynx, Surg., Gynec. & Obst. **76**:390-394 (April) 1943.

23. Orton, H. B.: Tumors of the Laryngopharynx, in Surgery of the Nose and Throat, edited by J. D. Kernan, New York, Thos. Nelson & Sons, 1942, pp. 527-535.

24. Schall, L. A., and Ayash, J. J.: Cancer of the Larynx: A Statistical Study, Ann. Otol. Rhin. & Laryng. **57**:377-386 (June) 1948. Ogura and Bello.[17] Orton.[19]

25. Garland, L. H.: Carcinoma of the Larynx: X-Ray Therapy, Laryngoscope **62**:75-89 (Jan.) 1952.

26. Cutler, M.: Radiotherapy of Early Cancer of the Larynx: Five Year Results in 156 Cases, J. A. M. A. **142**:957-963 (April 1) 1950.

CARCINOMA OF THE LARYNX AND TUBERCULOSIS

Included among the 116 patients with carcinoma of the larynx represented in this study are 6 patients with pulmonary tuberculosis. Our interest was aroused by the frequent gross similarities between tuberculous and malignant disease of the larynx, apparent in color photography. It soon became evident that almost any tuberculous lesion could be matched pictorially by one of proved carcinoma and vice versa.

A 48-year-old man with advanced pulmonary tuberculosis and a normal voice had noticed difficulty in swallowing for one month. Edema and ulceration of the left arytenoid (Fig. 12) subsided completely with the administration of streptomycin therapy.

A 55-year-old man had been aware of throat discomfort and hoarseness for two weeks. There was a tumor of the right aryepiglottic fold extending to the postcricoid area (Fig. 11), with cervical metastasis. Biopsy disclosed a squamous-cell carcinoma.

Aside from the gross similarity between the two conditions, the diagnostic aspects of carcinoma of the larynx and tuberculosis of the larynx may be considered under three headings: (1) coexistence of the two diseases in the same larynx, (2) laryngeal tuberculosis as a possible precursor of cancer, and (3) differential diagnosis of the two diseases.

Various observers have reported histologic proof of the presence of the two diseases in the same larynx. Jackson and Jackson[27] referred to a number of cases of cancer of the larynx associated with tuberculosis. In the 116 patients with cancer in this series and in over 200 patients with laryngeal tuberculosis observed by us, the two diseases were never found to coexist in the same larynx. The chief significance of the occasional presence of both tuberculosis and cancer of the larynx would seem to be a medical oddity, though there can be no doubt of their rare simultaneous occurrence.

The possible role of fibrous tissue from tuberculosis or syphilis of the larynx as a precursor of cancer has been described. There is little clinical evidence to support this relationship. Many of the 200 patients with tuberculous laryngitis were observed for long intervals after the administration of streptomycin therapy, and the effects of healing are being studied histologically. Thus far there has been no instance of carcinoma developing at the site of the laryngeal tuberculosis.

One standard medical school examination question in otolaryngology used to require a differential diagnosis of syphilis, tuberculosis, and cancer of the larynx. In our experience, the differential diagnosis usually rests between tuberculosis and cancer. It is well known that cancer of the larynx may occur at any age, but most commonly between the ages of 45 and 65. One of the most surprising alterations in the age incidence of any disease has been evident in pulmonary tuberculosis. Until recently it was considered a disease of adolescents and young adults. Today the third, fourth, and fifth decades are the years of greatest incidence.

Hoarseness is the most frequent symptom in both cancer and tuberculosis of the larynx. Pain is more likely to be present in tuberculosis than in carcinoma, but exceptions are so common that this is not an accurate differential point.

The gross pathologic alterations of infiltration, ulceration, and tumor are common in both diseases (Figs. 11 and 12). It might be expected that laryngeal tuberculosis

27. Jackson and Jackson,[10] p. 215.

would be diffuse and bilateral while laryngeal cancer would be localized and unilateral. We have found an exact reversal of this situation so frequently that this has not been a reliable differential point.

One important objective finding concerns mobility of the vocal cord. Fixation occurs relatively early with moderate involvement in carcinoma. Extensive infiltration and tumefaction of the arytenoids may be present in laryngeal tuberculosis without impairment of the motion of the cords.

The advent of antimicrobial agents effective in tuberculosis offers the possibility of a therapeutic test to aid in the diagnosis. A number of reports disclose that cancer was suspected only after the lesion failed to improve with the use of streptomycin or paraaminosalicylic acid.[28] This trial by the use of antibiotic therapy is not recommended because of the valuable time which may be lost if a malignant growth exists.

The introduction of streptomycin and other agents has also been important in the diagnosis, since now there is no hesitancy in taking a biopsy specimen in laryngeal tuberculosis. Formerly this might have been deferred for fear of ulceration and the possible deleterious effect on the laryngeal disease. The rapid healing effect of streptomycin in laryngeal tuberculosis removes any possible harmful effects of the biopsy.

The presumptive or clinical diagnosis of laryngeal tuberculosis may be established with a high degree of accuracy by the demonstration of the pulmonary disease and the appearance of the larynx. Similarly, ruling out the disease in the chest would eliminate a tuberculous laryngeal lesion. However, the possibilities of error are sufficiently great to warrant biopsy in all laryngeal lesions suspected of being either tuberculosis or cancer.

SUMMARY AND CONCLUSIONS

Some of the diagnostic problems encountered in a study of 116 patients with laryngeal carcinoma have been presented.

The terms "cordal," "endolaryngeal," "subglottic," and "extracordal" have been used to designate the four principal types of laryngeal carcinoma.

Hoarseness was the only symptom of cordal carcinoma and was present in each of the 31 patients in this category. The duration of this symptom before diagnosis was an average of seven months. The widespread use of antibiotics may be a responsible factor in delayed diagnosis of early laryngeal cancer.

Leucoplakia was evident in 19 of 31 patients (61%) with carcinoma of the vocal cord. Inasmuch as leucoplakia figures prominently as a precursor of cordal carcinoma and is observed most often in heavy smokers, excessive smoking may be regarded as a predisposing factor in cancer of the vocal cord.

Hoarseness was uniformly present in 18 patients with endolaryngeal carcinoma. Throat discomfort of varying degree was also experienced by six patients in this group.

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28. Phillips, S., and Larkin, J. C., Jr.: Treatment of Intrinsic Carcinoma of the Larynx in Patients with Pulmonary Tuberculosis, A. M. A. Arch. Otolaryng. **54**:624-631 (Dec.) 1951. von Fraenkel, P. H.: Oro-Laryngeal Tuberculosis: Report of a Case, U. S. Nav. M. Bull. **49**:536-541 (May-June) 1949. Helg, J. R.: Cordite tuberculeuse ou epithelioma, Rev. laryng. **70**:485-486 (Nov.-Dec.) 1949.

The diagnosis of subglottic carcinoma was often obscure until invasion of the vocal cord or narrowing of the airway occurred. The average duration of symptoms before diagnosis in cases of subglottic carcinoma was 13.4 months, an interval appreciably greater than in other forms of laryngeal carcinoma.

Extracordal (extrinsic) carcinoma developed silently or with minimal symptoms. The commonest symptom was some form of throat discomfort, present in 69% of the patients.

In the entire group of 58 patients with extracordal carcinoma, 20 (34.5%) were found to have cervical metastasis on admission.

Multiple primary carcinomas were found in 7 of the 116 patients in this study.

Tuberculosis of the larynx and carcinoma of the larynx often presented comparable gross appearences. Biopsy in a case of laryngeal tuberculosis is warranted when cancer is suspected.

ACUTE IODISM FOLLOWING USE OF CHLORIODIZED OIL (IODOCHLOROL) IN BRONCHOGRAPHY

Report of a Case

MARTIN ASCH, M.D.
LOS ANGELES

THE FOLLOWING case of acute iodism and bronchopneumonia subsequent to bronchography with chloriodized oil (Iodochlorol), a chlorinated iodized peanut oil, is presented as the first of its kind recorded in the literature. An iodized poppyseed oil (Lipiodol [Lafay]) has been used as a diagnostic agent in bronchography since 1922, when it was introduced by Sicard and Forestier.[1] Undesirable reactions, while uncommon, have been reported as a result of the use of this agent. These reactions may be divided into two classes. The first class constitutes several local phenomena resulting from the tissue response provoked by the instillation of the contrast substance. Bronchial asthma, local atelectasis, and massive collapse of the lung have been cited as occurring during bronchography or shortly after its completion.[2] Focal lipid granulomatosis arising two to four weeks after bronchography has been described by several investigators.[3] Microscopic examination, as well as experimentally induced lesions, indicates that such granulomatous lesions are generally produced as a result of the lipoidal fraction of the contrast agent.[4]

From the Medical Service, Veterans Administration Center, General Medical and Surgical Hospital.

1. Sicard, J. A., and Forestier, J.: Méthode generale d'exploration radiologique par l'huile iodée, Bull. et mém soc. méd hóp. Paris **46**:463, 1922.

2. Hansen, O. S.: Death from Asthma, Minnesota Med. **19**:445, 1936. MacDonald, I. G.: The Local and Constitutional Pathology of Bronchial Asthma, Ann. Int. Med. **6**:253, 1932. Jacobæus, H. C.; Selander, G., and Westermark, N.: A Study of Acute Massive Atelectatic Collapse of the Lung, Acta med. scandinav. **71**:439, 1929. Pinchin, A. J. S., and Morlock, H. V.: Acute Massive Collapse of the Lung Following Lipiodol Injection, Brit. M. J. **1**:930, 1931.

3. (*a*) Rabinovitch, J., and Lederer, M.: Lipoid Pneumonia, Arch. Path. **17**:160, 1934. (*b*) Wright, R. D.: Reaction of Pulmonary Tissue to Lipiodol, Am. J. Path. **11**:497, 1935. (*c*) Brody, H.: Focal Lipid Granulomatosis of the Lung Following Instillation of Iodized Poppyseed Oil, Arch. Path. **35**:744, 1943. (*d*) Storrs, R. P.; McDonald, J. R., and Good, C. A.: Lipoid Granuloma of the Lung Following Bronchography with Iodized Oil, J. Thoracic Surg. **18**:561, 1949. (*e*) Fortner, H. C., and Miles, J. S.: Iodized Poppyseed Oil Granuloma: Report of a Case, Arch. Path. **49**:447, 1950.

4. Fried, B. M., and Whitaker, L. R.: Intratracheal Injection of Iodized Oil: Experimental Studies, Arch. Int. Med. **40**:726, 1927. Pinkerton, H.: The Reaction to Oils and Fats in the Lung, Arch. Path. **5**:380, 1928. Brown, A. L.: The Fate of Iodized Oil (Lipiodol) in the Lungs, Surg., Gynec. & Obst. **46**:597, 1928. Gowar, F. J. S., and Gilmour, J. R.: Changes in Lung Following Injections of Iodized Oil into Trachea, Brit. J. Exper. Path. **22**:262, 1941. Chesterman, J. T.: Lipoid Pneumonia Due to Iodised Oil, J. Path. & Bact. **54**:385, 1942.

The second class of reactions constitutes the syndrome of acute iodism and is directly attributable to the iodine fraction of the radiopaque agent. The alarming systemic effects usually begin 6 to 24 hours after administration of the iodide and is generally similar in nature to the reactions produced by iodides administered by other routes. It is felt that the syndrome is a result of a peculiar idiosyncrasy on the part of the patient, rather than a true allergic phenomenon.[5] Scadding [6] reported a case of acute iodism after bronchography with iodized oil in a patient who had previously been exposed to this agent and, in addition, had been receiving potassium iodide medication for bronchiectasis, without mishap. Signs and symptoms consist of coryza, salivation, injection of the conjunctivae, nausea, vomiting, and diarrhea. Dermatological findings include angioneurotic and periorbital edema; urticaria, and acneform, vegetative, and bullous lesions. Hemorrhagic bullae and purpuric lesions involving both skin and mucous membranes occur frequently. Tenderness associated with edema of the salivary glands may produce the picture of "iodide mumps." Recovery usually ensues and is complete within one to three months. Twenty-one nonfatal and nine fatal cases of acute iodism after bronchography with iodized oil have been reported.[7] Five of the nonfatal cases occurred in patients with pulmonary tuberculosis.

Laboratory examination commonly reveals albuminuria, which is frequently associated with hematuria and red blood cell casts. In addition, iodine may often be found in the urine.[8] The hemogram shows no specific changes.

Chloriodized oil, a chlorinated-iodized peanut oil, is commonly used as a contrast agent in bronchography. The introduction of both chlorine and iodine at the unsaturated carbon atoms is claimed to result in a more stable compound than if iodine alone were used.[9] Chloriodized oil contains 27% iodine and 7.5% chlorine organically combined with a highly refined peanut oil. In 1949, Storrs and co-workers [3d] reported the first untoward effect following the use of this agent in bronchography. His case

5. Irwin, T. M.: Iodism, Ann. Otol. Rhin. & Laryng. **61**:408, 1952.

6. Scadding, J. C.: Acute Iodism Following Lipiodol Bronchography, Brit. M. J. **2**:1147, 1934.

7. (*a*) Carmichael, D. A.: Iodine Poisoning and Iodism from Lipiodol, Canad. M. A. J. **26**:319, 1932. (*b*) Goldstein, D. W.: Fatal Ioderma Following Injection of Iodized Oil for Pulmonary Diagnosis, J. A. M. A. **106**:1659, 1936. (*c*) Amberson, J. B., Jr., and Riggins, H. M.: Lipiodol in Bronchography: Its Disadvantages, Dangers, and Uses, Am. J. Roentgenol. **30**:727, 1933. (*d*) Mahon, G. S.: Reaction Following Bronchography with Iodized Oil, J. A. M. A. **130**:194, 1946. (*e*) White, B., and Bayliss, C. G.: Acute Iodism Following Lipiodol Bronchography, M. J. Australia **2**:23, 1937. (*f*) Plehn, C. J., in discussion on Anderson, W.: The Treatment of Bronchial Asthma by Intratracheal Injections of Iodized Oil, New York J. M. **36**:1151, 1936. (*g*) Schneider, L., and Segal, J.: Bronchography with Iodized Oil Not Without Danger: Report of a Case, Am. Rev. Tuberc. **35**:590, 1937. (*h*) Sheldon, F. B.: Long Pulmonary Retention of Lipiodol, Radiology **40**:403, 1943. (*i*) Kooperstein, S. I., and Bass, N. E.: Pulmonary Reaction Following Intrabronchial Instillation of Lipiodol in Bronchial Asthma, Am. J. Roentgenol. **56**:569, 1946. (*j*) Armand-Delille, P. F., and Moncrieff, A.: Lipiodol in the Diagnosis of Bronchiectasis, Brit. M. J. **2**:7, 1924. (*k*) Landau, W.: Die Röntgendiagnose der Bronchiektasie mittels Kontrastöl, Deutsche med. Wchnschr. **51**:1115, 1925. (*l*) Miller, J. A., and Eglee, E. P.: Bronchograms in the Study of Pulmonary Disease, Am. Rev. Tuberc. **16**:19, 1927. (*m*) Scadding,[6] (*n*) Footnote 2.

8. Footnote 7*d*, *e*, and *f*.

9. Iodochlorol: Radiopaque Agent, Searle Reference Manual No. 39, Chicago, G. D. Searle & Co., 1947.

was one of pulmonary lipoid granuloma, with a subsequent fatal termination. To date, this report is the only one recorded of an undesirable reaction following the use of chloriodized oil in bronchography.

REPORT OF CASE

E. R., a 53-year-old white engineer, was admitted to the hospital with the following history. Two days earlier he had undergone bronchography with iodized and chlorinated peanut oil (chloriodized oil). The patient gagged considerably during the procedure, and, although a fair amount of the drug was expectorated, some was apparently swallowed. Shortly thereafter he began to complain of dull pain in the left lateral chest over the third and fourth ribs. The patient noted that his right eye was "sore and red" the next morning, and localized plaques of edema appeared, involving the buttocks, forearms, and fingers. Generalized aching and malaise forced him to seek bed rest. A new sharper pain exaggerated by deep inspiration began in the left lateral chest over the seventh and eighth ribs at 4 a. m. on the day of admission. The patient sought hospitalization when hemoptysis appeared and his dyspnea increased.

The past history revealed that the patient was afflicted with a chronic necrotizing pyoderma of the buttocks, trunk, and lower extremities of 32 years' duration, with severe exacerbations during the past year; this was associated with a chronic glomerulonephritis.

Physical examination revealed the patient to be acutely ill, with a temperature of 100.4 F. Areas of angioneurotic edema and many new acneform lesions, some with necrotic centers, were generally distributed. The older ulcerated necrotic lesions which the patient had already had for 32 years had undergone a severe exacerbation. The right eye was virtually closed by periorbital edema, and the conjunctiva was moderately injected. The left tonsil was indurated and covered with a blood-streaked purulent exudate. The salivary glands were neither enlarged nor painful, and the anterior and posterior cervical lymph nodes were enlarged and tender. The breath sounds were normal, except for an area of coarse moist râles at the base of the left lung. Percussion of the left chest, although resonant, was painful.

On admission to the hospital, the hemogram showed a white blood cell count of 11,000, with neutrophiles 77%, segmented cells 13%, lymphocytes 8%, monocytes 1%, eosinophiles 1%, red blood cells 4,300,000; hemoglobin 14.2 gm. per 100 cc.; sedimentation rate 38 mm. in one hour (corrected).

Urinalysis gave the following results: albumin 2+, 10 to 12 white blood cells per high-power field, reaction for occult blood positive, many hyaline casts, many coarsely granular casts, occasionally finely granular casts. Bacterial culture grew nonhemolytic Staphylococcus albus. Blood urea nitrogen was 22 mg. per 100 cc.; creatinine was 2.2 mg. per 100 cc. Total serum iodine was 65 γ per 100 cc. A portable chest plate revealed a left basal pneumonitis; no significant residual contrast medium was evident. (The patient had on a previous occasion been found to have a mild intermittent albuminuria; however, the present level was definitely elevated.) A serological test for syphilis was negative. Subsequent roentgenological examinations of the chest demonstrated extension to the base of the right lung. Total serum iodine one week later was 35 and one month later 8 γ per 100 cc.

The pneumonic process resolved uneventfully on a regimen consisting of streptomycin and oxytetracycline (Terramycin) systemically, postural drainage, and oxygen by nasal tube. In addition to the systemic antibiotic, neomycin ointment was applied locally to the lesions, with subsequent improvement.

COMMENT

A close similarity exists between the findings in the case report presented and those to be observed in the syndrome of acute iodism. The occurrence of the signs and symptoms 24 hours after exposure to an iodide and associated with an elevated total serum iodine level permits a diagnosis of acute iodism to be made.

The possibility that the chlorine fraction of the chloriodized oil may have played a role in the acneform eruption was considered; however, these eruptions have

appeared only after the local contact with unstable chlorinated hydrocarbons.[10] Butler [10a] suggested that more than local contact was involved in the development of acneform lesions due to unstable chlorinated hydrocarbons. He considered inhalation of these compounds as an important etiological factor, but no evidence was presented. Jones and Alden [10c] stated that acneform eruptions were not caused by internal absorption. Acute intoxications by chlorine rarely occur, since suffocation and death by pulmonary edema ensue even in the presence of relatively small quantities of chlorine gas in the air. When chlorine or the unstable chlorinated hydrocarbons are ingested, the chlorine liberated readily combines with sodium to form innocuous sodium chloride.[10c]

The pathogenesis of the left basal pneumonitis followed by the bilateral bronchopneumonia is not so clear. The complete resolution of the pneumonic process within three weeks makes a presumptive diagnosis of lipoid granuloma formation unlikely. In the earlier reports the lipoid granulomas did not resolve spontaneously and were discovered either at surgery or post mortem.

The elevated blood iodine level was difficult to explain. The reference manual [9] accompanying chloriodized oil states "that Iodochlorol [8] is routinely tested by incubation at 140° F. for ten days, and even this rigorous test fails to disturb the stability of the chemical combination." No mention is made regarding the stability of this compound in the presence of the enzymes of the gastrointestinal tract. A possibility which has to be considered in this case is that a fair amount of the iodized oil was probably swallowed when the patient experienced gagging and difficulty during the bronchography procedure. After digestion by the gastrointestinal enzymes, iodine could have been liberated and absorbed into the circulation via the intestinal mucosa. This could explain the elevated serum iodine and the syndrome of acute iodism.

SUMMARY

A case of acute iodism following the use of chloriodized oil (Iodochlorol) in bronchography is presented as the first of its kind in the literature at this time.

10. (*a*) Butler, M. G.: Acneform Dermatosis Produced by Ortho (2 Chlorophenyl) Phenol Sodium and Tetra-Chlorphenol Sodium, Arch. Dermat. & Syph. **35**:251, 1937. (*b*) Schwartz, L., and Peck, M.: Occupational Acne, New York J. Med. **43**:1711, 1943. (*c*) Jones, J. W., and Alden, H. S.: An Acneform Dermatergosis, Arch. Dermat. & Syph. **33**:1022, 1936.

THE ALLERGIC LOAD

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AND
WALLACE RUBIN, M.D.
NEW ORLEANS

INSPIRED by the reports and teachings of Hansel and others in recent years, an ever-increasing number of otolaryngologists have incorporated into their practices the treatment of allergic diseases of the ears, nose, and throat. Many of these men have retained their original enthusiasm, but some have become dissatisfied with their results and have abandoned allergic therapy entirely. Undoubtedly, one important reason for this unfortunate development was a failure to realize that good allergic management demands a consideration of the patient as a whole. Another major reason may be that, in their preoccupation with the antigen-antibody relationship, they failed to take into consideration some of the other factors which may greatly influence or modify the allergic reaction. Therefore, it seemed to us that a discussion of the concept of the "allergic load" might be of value at this time. While this concept may not satisfy all scientific requirements, it will provide one with a simple and common-sense approach to some of the problems that arise when dealing with allergic patients.

The "allergic load" may be defined as the sum total of the factors which play a part in the production or modification of allergic symptoms in the allergic person. The factors may be either contributory or exciting ones.

The contributory factors are incapable of producing an allergic attack themselves, but they affect the person in such a way as to precipitate or aggravate allergic reactions. In many instances they act by adversely affecting capillary function. Since one of the main features of the allergic reaction is capillary dysfunction in the form of dilatation and increased permeability, it can be readily appreciated that their presence would serve to increase the allergic load. Among the contributory factors may be mentioned such mechanical irritants as dusts, chemical fumes, and pungent odors from various sources, infection, emotion, changes in the weather, barometric pressure changes, temperature changes, endocrine imbalance, physical and mental exhaustion, malnutrition, changes in mineral and lipid metabolism, etc.

The specific, or exciting, factors are, of course, the antigens or allergens. The allergic load may be increased as a result of either massive exposure to a single antigen or simultaneous exposure to a number of substances to which the person is sensitive.

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Examples of massive exposure are numerous. Ragweed pollen may not produce symptoms in a certain person until the count reaches 100. Persons sensitive to house dust may not have symptoms until they are massively exposed, as during house cleaning.

Unfortunately, most allergic persons are sensitive to more than one substance. In Chart 1 we have graphically portrayed the sensitivities of a hypothetical patient. It will be seen that he is sensitive to three common allergens and, in addition, is affected at times by changes in the weather. Massive exposure to any one of the antigens might, of course, produce symptoms. On the other hand, when exposure is minimal, it might require a combination of three or more antigens to be present simultaneously for the production of symptoms. This summation of effect to push the patient over the threshold of symptoms serves to explain, at least in part, some frequently encountered phenomena. For instance, it might be the reason why allergic patients can at one time eat with seeming impunity a food that will precipitate a crisis at another time—the load at that particular time is not heavy. This might also explain why certain foods have been found to cause symptoms in a given person only

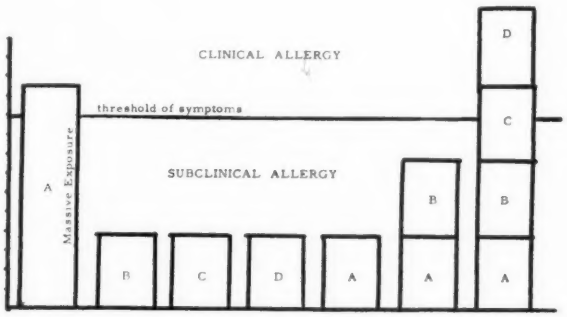

Chart 1.—Graphic representation of the allergic load. *A*, house dust; *B*, grass; *C*, egg; *D*, weather change.

during a particular season or when used in combination with other foods. An upper respiratory infection may produce asthma in a certain person at a time when the load is heavy but not at another time, when the load is light.

Many therapeutic measures are directed towards reducing the load. Included among these are procedures to make the dust-sensitive patient's home, particularly his bedroom, as dust-free as possible; campaigns to destroy ragweed during the ragweed season; the use of hypoallergenic cosmetics; trips to the seashore or mountains during certain seasons, etc. Theoretically, the object is to remove the allergen completely from the environment. Actually, this is seldom accomplished, for house dust is omnipresent; ragweed pollen blown by the wind from bushes hundreds of miles away contaminates the air, and the cosmetic-sensitive patient must mingle in society with persons who are not wearing hypoallergenic powder. In reality, what is accomplished is a reduction of the allergic load. With regard to the patient with multiple sensitivities, the therapeutic implication of the concept must be evident. To illustrate, let us return to the patient we previously considered who was sensitive to house dust, egg, and grass pollen and was adversely affected by sudden change in the weather. You will recall that we postulated that minimal exposure to any com-

bination of three of these factors at one time would result in symptoms. It follows, then, that this patient can be kept free of symptoms provided at least two of his sensitivities are well controlled.

So far, we have not discussed the threshold of symptoms in relation to the allergic load. Chart 2 represents a patient whose symptom threshold is low. This may be due to lowered resistance in the form of insufficient protective antibodies or, perhaps, to the presence of contributory factors such as were previously mentioned. Symptoms will be produced in such a case even though contact with the allergen or allergens seems relatively mild. In this connection it must be mentioned that a lowering

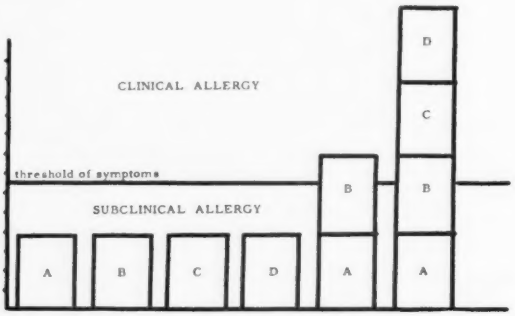

Chart 2.—Graphic representation of the allergic load; low threshold symptoms.

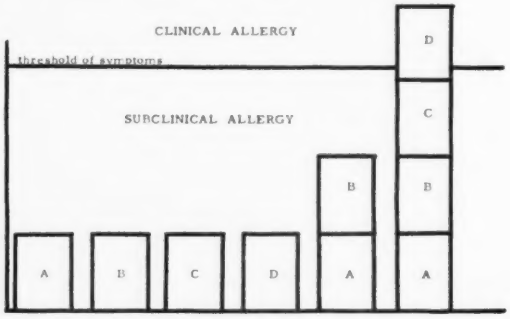

Chart 3.—Graphic representation of the allergic load; high threshold symptoms.

of the threshold of symptoms may be more apparent than real. This may be the case when the patient has numerous sensitivities or when he has a fairly high degree of sensitivity to a number of substances. Allergic management of this type of person is much more difficult than when the person has a high threshold of symptoms (Chart 3). In the latter case the degree of sensitivity may be low and or the number of sensitivities may be few. Factors causing a lowering of the threshold of symptoms may be thought of as increasing the allergic load, whereas those which lead to an elevation of symptom threshold decrease the load.

In the schematic representations it will be noticed that reference is made to a state of "subclinical allergy." This means that a person may be sensitive to one or

more substances which do not produce symptoms except under certain conditions of massive exposure or concomitant exposure with other allergens or when the symptom threshold is lowered by some contributory factor.

We should now like to proceed to a discussion of a few common conditions which we have found by experience to increase the allergic load and also to theorize as to the mechanism involved.

SMOKING

Besides being a local irritant and a possible allergen, tobacco smoke may add to the allergic load in other ways.

One cigarette contains 1 gm. of tobacco with 22 mg. of nicotine, one of the most toxic alkaloids known; 60 to 120 mg. of nicotine is lethal if given intravenously. Therefore, a person smoking one package of cigarettes daily will use 13,640 mg. of nicotine in a month. In the process he loads about 800 cc. of tar onto his mucous membranes.

Because of its sympathomimetic action, nicotine constricts the peripheral blood vessels and reduces the blood flow; it also first stimulates and then depresses the adrenal medulla and causes renal ischemia, with resultant inhibition of diuresis and retention of fluids.[1]

It should be obvious, then, that smoking will lower the threshold of symptoms and thereby increase the allergic load. In our opinion all allergic patients should give up smoking.

ANEMIA

It has been pointed out that in the allergic reaction the effectiveness of the circulation in the shock tissues is impaired and there is a decreased delivery of oxygen to the capillary walls and the tissues.

In anemia there is also a decreased delivery of oxygen, with resultant loss of capillary tone and increased capillary permeability.

One might conclude, therefore, that anemia will lower the threshold of symptoms in allergy, and such is actually the case. For this reason, any evidence of anemia in the allergic person merits attention.

HYPERCHOLESTEREMIA

An increase in the level of blood cholesterol is a common finding in the allergic patient. Measures taken to decrease the blood cholesterol levels often result in marked clinical improvement. Thus, hypercholesteremia apparently increases the allergic load.

Cholesterol enters the blood stream from either endogenous or exogenous sources. It is removed from the circulation by the Kupffer cells of the liver; the remaining cells of the reticuloendothelial system take very little part in the process normally. Should the Kupffer cells fail in their function or should there be increased mobilization of fat from the depots, the blood cholesterol rises. If hypercholesteremia persists, as it often does in allergy, all the various cells of the reticuloendothelial system become phagocytically active, engulf cholesterol from the circulation, and store it in their cytoplasm in order to relieve the blood stream of its excess cholesterol. When

1. Mibashan, J.: Smoking: A Clinico-Social Problem, Practitioner. **168**:282-288 (March) 1952.

cholesterol is deposited in the cell, it acts as a foreign body, and, if it remains for a sufficient length of time, it stimulates the proliferation of fibrous tissue.[2] From the standpoint of what is known of the allergic mechanism, the changes described may assume a great deal of importance, because protective antibodies are thought to be formed by the reticuloendothelial system.

SEX HORMONE INFLUENCES

Allergic diseases are often affected by sex hormone fluctuations. Before this relationship is discussed it might be well to review briefly the modern concept of female endocrinology.

The anterior lobe of the pituitary gland secretes three hormones which are concerned in this problem: (1) the follicle-stimulating hormone, which stimulates the immature ovarian follicle to grow during the first half of the menstrual cycle and to produce estrogen, the latter causing the endometrium to proliferate; (2) the luteinizing hormone, which causes the corpus luteum to secrete progesterone, which, in turn, causes the endometrium to become secretory; (3) the luteotrophic hormone, which causes the corpus luteum to be maintained in an active state.

When the blood estrogens and progesterone levels become too high, the anterior pituitary lobe is depressed and the blood levels fall. This causes menstruation. If conception and implantation occur, the premenstrual drop of estrogen and progesterone does not occur, and these hormone levels continue to rise, owing, in part at least, to the addition of chorionic hormones.

At the menopause the ovaries are no longer responsive to the follicle-stimulating hormone stimulation, and estrogen is not produced. Since the anterior pituitary lobe is not inhibited, the follicle-stimulating hormone level rises and produces the symptoms.

Certain patients have exacerbations during the premenstrual period, when the estrogen and progesterone levels are high. These hormones cause vasodilatation, salt retention, and extracellular edema, all of which increase the allergic load.

As Hartman [3] points out, these patients become worse during pregnancy and are relieved at the menopause. Treatment consists of combatting the estrogen excess with methyltestosterone given orally during the postovulatory and premenstrual phase.

In contradistinction to the above type of patient are those who have an exacerbation of allergic difficulties during the first day or two of menstruation. At this time blood estrogen, progesterone, and follicle-stimulating hormone levels are low. They improve during pregnancy and become worse at the menopause. The mechanism here is by no means clear, but it is felt that a type of withdrawal phenomenon lowers the allergic threshold. These patients are treated by administering a large dose of an estrogenic substances just before or during the menstrual phase and so timed as to prevent or buffer the exaggerated drop in blood estrogen.

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2. Stambul, J.: Mechanisms of Disease: A Study of the Autonomic Nervous System, the Endocrine System, and the Electrolytes in Their Relationships to Clinical Medicine, New York, Froben Press, Inc., 1952.

3. Hartman, M. M.: The Use of Sex Hormones in Allergic Disorders, Ann. Allergy **5**:467-477 (Sept.-Oct.) 1947.

HYPOTHYROIDISM

Christman [4] is of the opinion that hypothyroidism of a mild degree is commoner than is generally thought. He found 138 cases of mild hypothyroidism in 1,000 consecutive unselected cases in his internal medical practice, an incidence of 13.8%. Hueper [5] points out that hypothyroidism causes stagnant anoxia and increased permeability of the capillary wall through excessive slowing of the blood flow. Since this is precisely what is thought to occur in the allergic state, it seems safe to assume that this condition increases the allergic load.

PSYCHOSOMATIC INFLUENCES

Most of us have been impressed at some time with the important part played by psychic factors in either initiating or maintaining allergic diseases.

All theories regarding the etiology of psychoneuroses assume some constitutional or hereditary factor as a background.[6] As far as we are concerned in this presentation, one of the most important of these is the inherited response pattern of the autonomic nervous system. Generally there is increased activity of the branches of the autonomic nervous system during emotional reactions. However, some persons manifest a preponderance of one part of the system. It is felt that such autonomic imbalance is, at least in part, genetically determined,[7] a physiologic background which determines certain aspects of personality and behavior. As compared with persons with sympathetic preponderance, those with parasympathetic preponderance have lower basal metabolic rates and systolic blood pressure and more emotional inhibition; show less emotional intensity, muscular tension, fatigability, and frequency of activity; are more dominant; come from child-centered homes with dominant mothers, and manifest palmar sweating, with a very narrow differential between the palmar and skin temperature.

It is not difficult to see how the presence of such an autonomic response pattern would increase the allergic load.

SUMMARY

A quantitative concept of the allergic reaction and some of the factors affecting it are discussed. This concept may be termed "the allergic load."

The "allergic load" is the sum total of the factors which play a part in the production or modification of allergic symptoms in the allergic person.

Therapeutic and other implications of the concept are considered.

A few of the conditions which increase the allergic load are discussed.

4. Christman, H. E.: Milk Hypothyroidism: A Common Disease, Ohio M. J. **46**:965-970 (Oct.) 1950.

5. Hueper, W. C.: Etiology and Causative Mechanism of Arteriosclerosis and Atheromatosis, Medicine. **20**:397-442 (Dec.) 1941.

6. Bauer, J., Constitution and Disease: Applied Constitutional Pathology, New York, Grune & Stratton, Inc., 1945.

7. Wenger, W.: Preliminary Study of Significance of Measures of Autonomic Balance, Psychosom. Med. **9**:301-309 (Sept.-Oct.) 1947.

WARTIME EXPERIENCE WITH SKULL INJURY AND BLAST AND THEIR EFFECT UPON THE EAR

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I. HISTORICAL DATA

MILITARY acoustic trauma caused by gunpowder explosions has probably been with us since 1353 A. D., when Typsiles of Augsburg introduced gunpowder to Germany, thus inaugurating its use by the people of western Europe.[1] Several centuries were to elapse, however, before efficiently operating explosive weapons completely replaced the battering ram, the long bow, and the sword.

In 1886, one who signed himself "Volunteer" brought the problem to the attention of the readers of the *Lancet*. He asked, "Can any of your readers suggest a remedy for deafness caused by rifle shooting?" The answer, presented by John Chippendale in a later issue, was, "Allow me to suggest the wearing of cotton in the ears while at rifle practice. This is done in France, at any rate, by artillerists to prevent deafness and bleeding of the ears." [2]

A volunteer of a more recent date has testified to the perseverance of the problem. In "Mr. President" Harry S. Truman describes a World War I incident in which "a French 155 mm. battery fired over my head, and I still have trouble hearing what goes on when there is a noise." [3]

From the period of the *Lancet* interchange to the present time, otologic literature has contained numerous reports on acoustic trauma, especially during and after World War I, when there was a large incidence of auditory defects caused by exposure to extraordinary conditions of excessive noise. In 1907, Witmaack attempted to prove that bone conduction is the pathway of intense noise traumatizing the internal ear, involving the spiral ganglia, nerve fibers, and organ of Corti.[4] This contention was disproved later by von Eicken (1909), Hoessli (1912), and Siebenmann (1915), all of whom, by different methods, proved that air conduction was the pathway.[4]

Hoessli [5] had two groups of guinea pigs. In one group the incus was removed.

Presented as a candidate's thesis in partial fulfilment of the requirements for membership in the American Laryngological, Rhinological and Otological Society, Inc.

1. Braun, K.: Short Historical Sketch of Gunpowder, translated by J. P. Wisser, New York, D. Van Nostrand Company, Inc.

2. Bunch, C. C.: Symposium: Neural Mechanism of Hearing, Laryngoscope **47**:615-691 (Sept.) 1937.

3. Mr. President, edited by W. Hillman, New York, Farrar, Straus & Young, Inc., 1952.

4. Guild, S. R.: The Otopathies of War, with Especial Reference to the Trauma Due to Air Waves, Bull. Internat. A. M. Mus. **7**:225-245, (May) 1918.

5. Hoessli, H.: Weitere experimentelle Studien uber die akustische Schädigung des Säugetierlabyrinths, Ztschr. Ohrenh. **64**:101-145, 1912.

The ossicles were not disturbed in the other. Both groups were then placed on the inside of a pipe which had machine hammers beating upon it for varying periods so that air- and bone-conduction factors were simultaneously present. On microscopic examination the animals operated on showed normal cochleae. Those in which the ossicular chain had not been broken showed degenerative changes in the cochleae. In later work, Prenant and Castex,[6] in 1916, subjected rabbits and guinea pigs to artillery fire at Fontainebleau, France. They found the organ of Corti disorganized principally in the first and second turns of the cochlea, and in some cases they found hemorrhage, atrophy, and shrinking of the nerve structure. In all instances, the vestibular labyrinth remained intact.

During World War I, "noise deafness" and "explosion deafness" were commonly seen in the field hospitals, and many clinical reports appeared in the literature. J. S. and John Fraser[7] examined four temporal bones from soldiers who died after exposure to "severe concussion" (blast). They found the tympanic membrane ruptured. There was hemorrhage in the middle ear and very slight hemorrhage in the scala tympani in the region of the round window and in the basal coil of the cochlea; hemorrhage was also present at the fundus of the internal acoustic meatus. The nervous elements showed degenerative changes, and the organ of Corti had a "ghost-like appearance." The structures of the membranous labyrinth appeared normal.

In the World War I period, discussion regarding acoustic trauma was mainly concerned with the desire of determining a working clinical analysis. The Frasers stated that it was difficult to draw a line between "noise" deafness and "shell" or "explosion" deafness, because both conditions are probably due to excessive movements of the atmosphere converged to the labyrinth. Captain J. H. Marriage,[8] in discussing prognosis, stated that he knew of no means of telling whether hearing would be recovered. He believed that if marked improvement did not take place within six to eight weeks the outlook would be extremely bad. He also found that tinnitus often persisted when hearing returned. C. E. West[9] had a similar experience in dealing with glancing scalp wounds when the pericranium alone was involved. He also stated that he was "hopelessly in the dark" with regard to prognosis of the gross perception deafness that occurred in such cases.

Stacy Guild,[10] writing on war deafness in 1941, stated that little that was new had been written about acoustic trauma since World War I. He compiled a complete bibliography of 177 papers published previous to 1918; 160 of these were written in the period from 1914 to 1917.

In Guild's complete report, "The Otopathies of War, with Especial Reference to the Trauma Due to Air Waves,"[4] he cites an informative, careful and time-tested study done by Yoshii[11] at Basel, Switzerland, in 1909. Histologic studies were done

6. Prenant, and Castex, A.: Recherches expérimentales et histologiques sur la la commotion des labyrinthes (surdités de guerre), Paris méd. **23**:197, 1917.

7. Fraser, J. S., and Fraser, J.: Morbid Anatomy of War Injuries of the Ear, Proc. Roy. Soc. Med. **10**:59-90, 1917.

8. Marriage, H. J.: Special Discussion on Warfare Injuries and Neuroses, Proc. Roy. Soc. Med. **10**:54, 1917.

9. West, C. E., in discussion on Fraser and Fraser,[7] p. 92.

10. Guild, S. R.: War Deafness and Its Prevention, Ann. Otol. Rhin. & Laryng. **50**:70-76 (March) 1941.

11. Yoshii, U.: Experimentelle Untersuchungen über die Schädigung des Gehörorgans durch Schalleinwirkung, Ztschr. Ohrenh. **58**:201-251, 1909.

on guinea pigs to determine the effect on the inner ear of detonation caused by a revolver fired near the animals. Two series of guinea pigs were used. One group was subjected to one detonation and the other to daily detonations. Yoshii stated:

Those which were subjected to one detonation show pathologic changes in the Organ of Corti. The outer and inner hair cells are swollen and loosened from their support. The nuclei are pushed upward and broken into small fragments; Deiter cells lose their structure and become changed into a homogeneous mass. Their supporting fibrils perish. The pillars remain but become bent and appear only indistinctly. The narrowed tunnel space is filled with a homogeneous mass, which stains faintly with eosin, hence appears like extravasated cell contents. Nuel's space and the outer tunnel space are no longer present; the cells of Hensen are deformed and flattened out. Frequently a great space is found between the loosened hair cells and the curve of the cells of Hensen.

These above changes are all found immediately after the first detonation and reach their height after two or three days. Then a regeneration process sets in slowly. Thus the Organ of Corti is found already somewhat improved in the animals killed eight days after the single detonation. The bent pillars straighten out and are recognized clearly. The hyaline mass in the tunnel is absorbed and the space itself restored. The hair cells, Deiter cells and their supporting fibrils are not clearly differentiated in this stage of repair. The healing process does not proceed evenly in all turns. While the basal turn is showing an improved condition, the other turns still show evident disturbances.

In after days, the regenerative process advances; the cells of the sulcus spiralis externus and internus and the cells of Hensen take their previous form. The restoration of the hair cells and Deiter cells seems to occur very slowly and incompletely because the animals killed 60 days after the single detonation showed that only in the basal turn were the hair cells and the Deiter cells in an improved but not fully normal state. In the remaining turns clearly differentiated hair cells and Deiter cells cannot be demonstrated. In the daily detonation animals, the degeneration *proceeded* until Corti's Organ was replaced by a flat epithelium as in any noise deafness, and as in animals exposed for a long time to a single tone.

This description is quoted from Dr. Guild's translation. These studies were repeated as recently as 1950 by Krejci and Bornschein,[12] but with the addition of electrocochleogram potentials. Their guinea pigs showed a dip between 3500 and 4500 cps. Their vital fixations, after 48 hours, showed degeneration similar to those described in the external air cells and Deiter cells from the end of the first turn to the middle of the second turn. When the pistol shots were repeated the changes progressed to the "ghost-like appearance" described by Frasers and by Yoshii.

Biochemical and biophysical studies have been done on the spiral-ganglion cells of animals exposed to measured-sound pressures and measured durations of exposure. These studies of Hamburger and Hyden [13] on the alterations in the nuclei of the ganglion cells led them to the following conclusions: 1. The intensity of and exposure time to a pure tone are directly related to the depletion of protein components in the cell nucleus and nucleolus as compared with the normal values for unexposed ears. 2. Animals surviving 21 days after exposure had normal concentration. 3. Animals killed in intervening periods had concentrations varying with the time elapsed after the exposure.

All the studies cited are of considerable importance and provide the pathogenetic explanations of the clinical recovery from temporary hearing impairment following exposure to muzzle blast and gunfire.

12. Krejci, F., and Bornschein, H.: Knalltrauma und c^5'-Senke beim Meerschweinchen (Bioelektrische und histologische Untersuchungen), Pract. oto-rhino-laryng. **12**:1-8, 1950.

13. Hamburger, C. A., and Hyden, H.: Cytochemical Changes in the Cochlear Ganglion Caused by Acoustic Stimulation and Trauma, Acta oto-laryng., Supp. 61, p. 189, 1945.

The use of loud sounds together with electropotentials in studies on animals have provided observations that shed considerable light upon the difference in function of the external and internal hair cells. These and other observations were made by means of histologic study of the degenerative changes resulting from measured-noise trauma. The information gathered has been correlated with deafness studies on humans subjected to prolonged exposure to loud and fatiguing tones. Davis and associates [14] found that the greatest hearing loss after exposure to a high-pitched fatiguing tone occurs about one-half an octave above the exposure tone and not below the frequency of this tone.

The high incidence of acoustic trauma again engaged the attention of military otologists in the recent war, and among the numerous recent reports in the literature are those of McReynolds,[15] Silcox and Schenck,[16] Ireland,[17] Korkis,[18] Collier,[19] and Collins.[20] Rüedi and Furrer,[21] in 1947, published a most scholarly monograph encompassing the pathologic anatomy, pathogenesis, and clinical and prophylactic aspects of acoustic trauma. Reference is made to many of those authors in the following report.

II. DATA AND CRITERIA FOR STUDY

From September, 1944, to October, 1945, I had the opportunity of directing the otolaryngologic examinations of about 29,000 military personnel at the Wakeman Army Medical Center at Camp Atterbury, Ind.

It was from this large group of soldiers that the data for this study were obtained. Any soldier with a history indicating that he had been exposed to and disturbed by (1) extraordinary conditions of excessively loud noise or (2) nearby blasts of explosives or had sustained (3) a direct head injury was given the questionnaire illustrated in Figure 1. He was assisted by one of two nurses on duty, as well as by the audiometrist, in arriving at the appropriate answers if he found any of the questions obscure. In this way, the probability of accuracy was enhanced.

In addition to a complete otolaryngologic examination, each patient who indicated exposure to any of the situations listed was tested by air conduction on an accurately calibrated Maico D-5 audiometer. The audiograms were taken by a highly competent sergeant who was painstakingly trained by me to a strict discipline of accuracy in testing. This assistant, by virtue of his patience and interest in the project, assured the professional staff of accurate audiometry. Both spoken-voice

14. Davis, H. S.; Morgan, C. T.; Galambos, R., and Smith, F. W.: Report on Temporary Deafness Following Exposure to Loud Tones and Noise, Medical Research, O.S.R.D., United States Department of Commerce, Sept. 30, 1943.

15. McReynolds, G. S.; Guilford, F. R., and Chase, G. R.: Blast Injuries to the Ear: Texas City Disaster, Arch. Otolaryng. **50**:1-8 (July) 1949.

16. Silcox, L. E., and Schenck, H. P.: Blast Injury of the Ears, Arch. Otolaryng. **39**:413-420 (May) 1944.

17. Ireland, P. E.: Traumatic Perforations of Tympanic Membrane Due to Blast Injury, Canad. M. A. J. **54**:256-258 (March) 1946.

18. Korkis, F. B.: Effect of Blast on Human Ear, Brit. M. J. **1**:198-201 (Feb. 9) 1946.

19. Collier, D. J.: Injuries to the Ear in War Time, M. Press **207**:337-338 (May 27) 1942.

20. Collins, E. G.: Aural Trauma Caused by Gunfire, J. Laryng. & Otol. **62**:358-390 (June) 1948.

21. Rüedi, L., and Furrer, W.: Das akustische Trauma, Monograph, Basel, Switzerland, S. Karger, 1947.

Answer These Questions Carefully

WARD NO._____ DATE_____ 19___

NAME_____ GRADE_____ ASN_____ AGE_____
(Last) (First) (Middle)

ARM OF SERVICE_____ LENGTH OF SERVICE_____

HOME ADDRESS_____
(Street) (City) State)

HOW LONG HAVE YOU BEEN HARD OF HEARING?_____

DID YOU EVER HAVE DISCHARGING EARS?_____ WHICH ONE?_____

HEADACHES?_____ WHAT PART OF HEAD?_____ DIZZINESS?_____

EARACHES?_____ VOMITING?_____ RINGING IN EARS?_____

RING SOUNDS LIKE WHAT?_____

DATE IF YOU HAD: MALARIA?_____ MEASLES?_____ MUMPS?_____
CHICKEN POX?_____ SMALL POX?_____ WHOOPING COUGH?_____
SCARLET FEVER?_____ DIPHTHERIA?_____ PNEUMONIA?_____
TYPHOID FEVER?_____ DID YOU EVER HAVE A HEAD INJURY?_____

DESCRIBE_____

HAVE YOU EVER HAD ANY OPERATIONS (PARTICULARLY EAR, NOSE OR THROAT)_____

HOW MANY COLDS IN ONE YEAR DO YOU HAVE?_____ EVER HAVE ANY HAY FEVER_____ ALLERGY_____

WERE YOU OVERSEAS, IF SO, WHERE AND HOW LONG?_____
WERE YOU UNDER FIRE, IF SO, WHAT KIND? DESCRIBE HOW IT AFFECTED YOU_____

DID YOU EVER TAKE ATABRINE OR QUININE OR OTHER DRUGS? IF SO, HOW MANY PILLS DAILY AND EFFECTS?_____

ARE ANY OTHERS IN YOUR FAMILY HARD OF HEARING? (INCLUDE UNCLES, AUNTS AND GRANDPARENTS)_____

HOW DO VOICES SOUND TO YOU? CLEAR?_____ JUMBLED?_____ NONE?_____

LOUDER IN NOISY PLACES?_____ DISTANT IN NOISY PLACES_____

HAVE YOU EVER HAD TREATMENT TO YOUR EARS?_____ DESCRIBE_____

DID YOU HAVE CONCUSSION OR A SKULL INJURY?_____

WHEN (MONTH, DAY, YEAR AND TIME OF DAY)?_____

WHERE?_____

ARE YOU IMPROVED?_____ DESCRIBE:_____

HAVE YOU HAD TREATMENT FOR YOUR EAR CONDITION?_____

DESCRIBE:_____

WHAT CALIBER BULLET OR EXPLOSIVE WERE YOU EXPOSED TO?_____

IF YOU WERE INJURED, DESCRIBE THE TYPE OF INJURY:_____

(Use back of sheet for details)

Fig. 1.—Questionnaire answered by each patient studied.

tests and the audiogram were taken in a soundproof room. The clinic was on the fringe of the camp, which in turn was situated in a farm area of southern Indiana, 30 miles away from any trace of industrial noise.

The mean age of the patients (all men) was 27 years. The youngest was 18 and the oldest 52.

Of the approximately 29,000 soldiers examined, there were 490 who offered a history of exposure to light or heavy gunfire with or without direct head injury and who fulfilled all the criteria listed hereafter.

Qualifications

Any patient who could not fulfill the seven criteria listed was considered unsuitable for study. These criteria were:

1. A history of normal hearing bilaterally before military service.

2. A completely negative family history of deafness unless the family history was carefully qualified with a detailed history of acquired otologic disease or degeneration.

3. No quinine medication either before or during military service.

4. A negative otologic history with regard to recurrent colds and the exanthemata of childhood.

5. A negative history of otorrhea or evidence of healed dry perforations of either drum before exposure to military otologic injury.

6. No evidence of malingering, hysteria, or psychic deafness.

7. A negative history of scrub typhus. (Two epidemics in 1944 in North Burma and Malaya caused a high incidence of perception deafness in recovered patients.)

Hearing Losses

Each air-conduction audiogram was classified, for our study, according to Carhart's [22] scheme of pure-tone audiogram tracing trends. This classification was useful in the recognition of the high incidence of what Carhart describes as "notching" and abrupt drops in hearing beyond the 1500-cps range. His classification is not included in this paper, because it is not universally known or used. Graphs of each individual audiogram, although ideal for accuracy, are too numerous and unwieldy and would leave no usable impression of the gradient that is present in each type of acoustic injury. We therefore elected to present in graphic form the combined findings of each type. It is important to understand before studying the graphs illustrated that the curves must not be considered as, or confused with, a standard type of curve for each classification of acoustic trauma. It is a gradient, or a pattern in our cases. The trend of pure-tone loss for each octave was determined by adding the decibel losses and dividing the sum by the number of cases in each group. The figures thus obtained were plotted on the audiogram charts.

About three years ago a follow-up study was attempted. A trial mailed request was made of 25 random patients for information about their hearing, but no replies were received. Possibly some of these patients did not reply because they feared that disability compensation might be affected. At a later date, perhaps with cooperation of the appropriate government agency, it may be possible to eliminate this fear and other factors interfering with replies and to obtain some follow-up information. The home addresses of all the patients are in my files.

22. Carhart, R.: An Improved Method for Classifying Audiograms, Laryngoscope **55**:640-662 (Nov.) 1945.

Fig. 2.—Preoperative view (*A*) and postoperative view (*B*) of M. M., infantry private aged 26, who received gunshot wound behind right eye, with development of arteriovenous aneurysm and shrapnel injury in right postauricular region, with atresia.

Fig. 3.—Preoperative view (*A*) and postoperative view (*B*) of C. B., private in Signal Corps, who received shrapnel injury of left external auditory canal, zygoma, and cheek, with development of atresia of left external auditory canal.

III. ATRESIA OF THE EXTERNAL AUDITORY CANAL DUE TO INJURY

In spite of the new type of helmet of World War II, injuries to the auricle were not uncommon. In many instances of penetrating ear injury, there was associated atresia of the canal in varying degrees and depths, depending upon the deflection of the missile causing the injury. Many soldiers survived extensive destruction of the base of the petrous pyramid. Peripheral facial paralysis on the side of injury was a frequent concomitant.

Our treatment of atresia of the canal secondary to injury gave us an opportunity to utilize the endaural No. 1 and No. 2 incisions of Lempert.[23] These incisions were particularly effective in this condition because they allowed a conservative operation, preserving the skin of the posterior canal wall as well as limiting the the amount of mastoid bone to be removed. The external auditory canal was enlarged by curette and gouge (electric burr was not available) at the expense of the exposed posterior bony external canal. The most common atresia was that of a funneling rosette type of scar. After careful discission of the recent scars with a cataract knife, the bony canal having been previously enlarged, the auditory canal was generally restored to satisfactory patency. With the cooperation of the

TABLE 1.—*Causes of Head Injury in One Hundred and Six Soldiers*

High-speed missiles		Contact blows	
Heavy artillery	35	Kicked on head	1
Mortar fire	13	Struck by board	1
Various projectiles	11	Struck by bottle	2
Rifle fire	9	Struck by baseball	1
Grenade fragments	9	Truck overturned	1
Mines	7	Knife wound	1
Bombs	6	Bullet grazed scalp	1
Machine-gun fire	5		
Dynamite	3		

dental prosthetic unit, lucite ear moulds were procured and used as obturators, and when these were not available, dental compound moulds were utilized. Thin split-skin grafts were occasionally necessary for long scars involving large losses of healthy canal-wall skin. These were maintained in position effectively by draping the graft, raw surface out, over the dental compound and permitting it to remain undisturbed for about 10 days (Figs. 2 and 3). I do not recall any "nontakes" with this procedure, even in the presence of slight middle-ear discharge. Occasionally, bits of cartilage of the anterior edge of the cavum conchae and anthelix had to be sacrificed to break the cartilaginous spring to avoid secondary narrowing contracture. In a few instances, when the floor of the canal was found to be excessively scarred and thickened, the external opening of the auditory canal was somewhat changed from its normal position toward a higher level at the junction of the anthelix and the tragus (Fig. 3).

IV. DIRECT INJURY TO THE SKULL

We observed 106 soldiers who had recently sustained a direct blow to the head. These men were subjected to head injury from the causes shown in Table 1. Eighty-six sustained penetrating fractures of the skull. Table 2 shows the site of contact of the missile causing the fracture.

23. Lempert, J.: Lempert Fenestra Nov-Ovalis with Mobile Stopple, Arch. Otolaryng. **41**:1-41 (Jan.) 1945.

Table 3 shows the causative agents of the fractures involving the temporal and vault regions.

About 75% of these men sustained skull fractures from high-speed missiles, generally shrapnel and bullets. It was natural to find that the temporal region was the commonest site of fracture in soldiers with ear complaints. The incidence was 40%.

TABLE 2.—*Site of Skull Fracture in Eighty-Six Soldiers*

No. of Patients	Site of Fracture
19	Temporal right
16	Temporal left
2	Temporal left and mandible
1	Temporal left and frontal
1	Temporal right and frontal
1	Temporal bilateral and right orbit
18	Vault
2	Occipital
4	Occipital left
1	Occipital right and left
1	Occipital left and frontal
1	Frontal center
2	Frontal left
2	Frontal right
1	Neck left
1	Neck right
2	Mandible left
2	Mandible right
3	Face front
2	Face left
4	Face right
86	

TABLE 3.—*Fractures Involving Temporal and Vault Regions*

Right temporal region, average age of patient 27	
Shrapnel	14
Bullet	3
Bomb fragment	1
Unknown	1
Left temporal region, average age of patient 26	
Shrapnel	12
Bullet	3
Truck overturned	1
Vault region, average age of patient 26	
Shrapnel	18

The composite audiograms of the fractures of the right and left temporal regions (Figs. 4 and 5) demonstrate profound losses of between 40 and 50 db. in the conversational-tone spectrum. The slightly better hearing in the left as compared with the right ear emphasizes that these audiograms show composite-trend curves indicating the degree of hearing loss of the involved side, as well as illustrating the relative freedom from involvement in the contralateral ear. In this group 11 patients indicated in the questionnaire that they believed that both ears had depressed hearing, and 18 complained of bilateral tinnitus. These symptoms are corroborated by the audiograms, for we observed that there is a progressive downward trend beyond

2048 cps for the ear on the side of the clinically uninvolved temporal bone. A comparative evaluation using this ear as a control is not possible, because many of the soldiers were exposed to repeated loud sounds from artillery and gunfire for varying periods before being put out of action by the fracture sustained. The previous exposure, I believe, adequately accounts for the contralateral tinnitus and mild deafness encountered (Appendices I and II). The following case history is

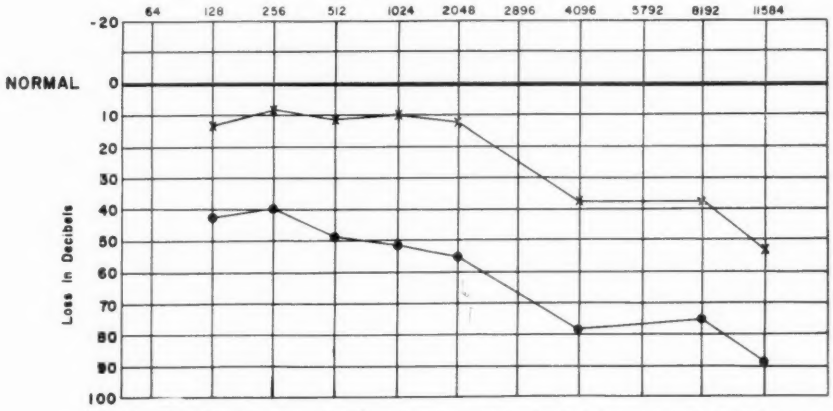

Fig. 4.—Trend audiogram for 19 patients who sustained right temporal skull fracture. Air-conduction curves, with dot for right ear and *x* for left.

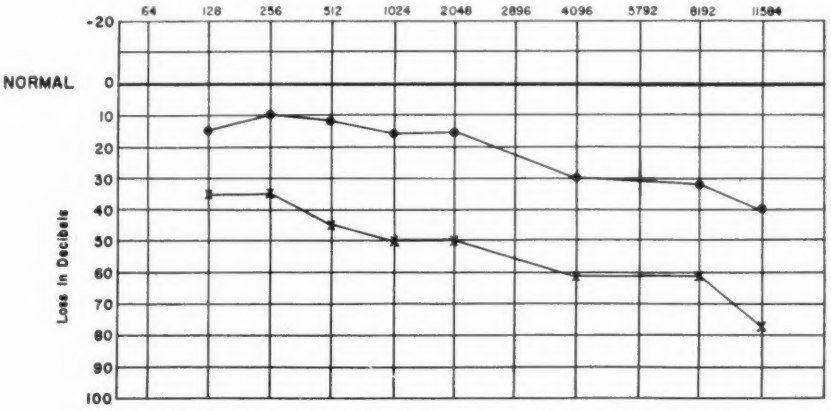

Fig. 5.—Trend audiogram for 16 patients who sustained left temporal skull fracture. Air-conduction curves, with dot for right ear and *x* for left.

presented to illustrate blast trauma and fracture accounting for the bilateral audiogram threshold elevation.

A 25-year-old infantry sergeant, L. H. B., in command of a 90-mm. gun, was struck by a projectile in the left temporal region. He was semiconscious for 36 hours. His previous military service consisted of three years of active duty as a 90-mm.-gun commander in Panama and the South Pacific. His audiogram, as illustrated, shows a perception type of hearing loss due to loud noise in the right ear and a mixed type of loss on the injured left side. The conversational hearing in his right ear was 15 ft. (457 cm.) and in his left ear 5 ft. (152 cm.) (Fig. 6).

Of the 35 victims of temporal-region fracture, 34 indicated at least unilateral defective hearing on the questionnaire. The one who did not was a private, E. L. C., 32 years old, in the armored infantry. He was struck on the left parietotemporal region by shrapnel at Boxmeer, the Netherlands, on Oct. 3, 1944. The defect required a tantalum graft. Although not complaining of hearing loss, he was annoyed by bilateral tinnitus, which seems consistent with the high-tone defect in his audiograms (Fig. 7).

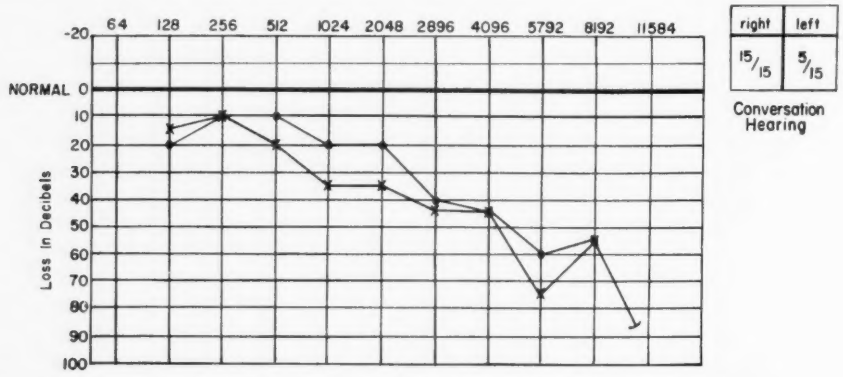

Fig. 6.—Audiogram for L. H. B., aged 25, struck by projectile in left temporal region. Air-conduction curves, with dot for right ear and *x* for left.

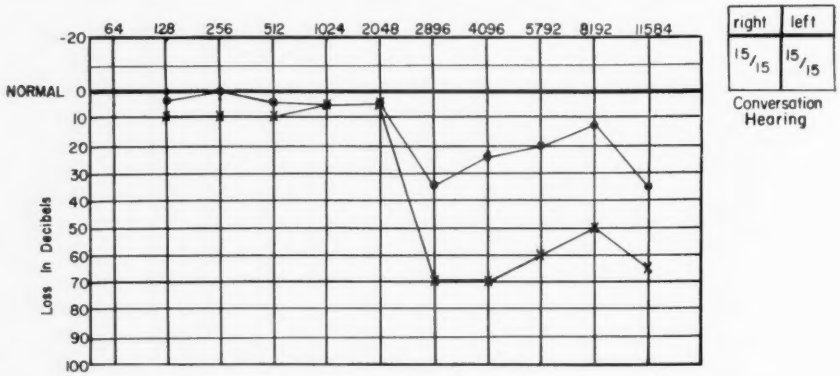

Fig. 7.—Audiogram for E. L. C., aged 32, struck by shrapnel in left temporal region. Air-conduction curves, with dot for right ear and *x* for left.

Tinnitus, the symptom so commonly seen in acoustic trauma, was an almost universal complaint in the fracture cases. This symptom was persistent in 85% of these cases 9 to 10 months after the injury was incurred. There was no instance in which the tinnitus was localized solely to the ear opposite the site of the injury.

Tinnitus often is as perplexing to the otologist from the viewpoint of diagnosis and therapy as it is annoying to the victim. I was hampered by therapeutic limitations when confronted with this persistent symptom following blast and direct-blow trauma. These limitations were particularly obvious because, as a rule, the patient

was far more concerned with, and desirous of therapy to end, the "buzzing" than with the prognosis in regard to his hearing impairment, even when a profound functional hearing loss was present.

Following a suggested line of treatment, I administered vitamin A therapy to eight soldiers with persistent tinnitus, giving 100,000 units orally a day in divided doses. The suggestion came from R. Schillinger,[24] of Chicago, who stated that he had favorable results with tinnitus and hearing loss using this therapy in civilian practice. After a trial of three to four weeks my co-workers and I concluded that this treatment did not favorably influence the tinnitus or hearing loss in any of our cases.

Although the civilian post-office addresses of the subjects were indicated on the questionnaires for the sake of future follow-up, it was evident merely from the description of the character of the tinnitus which of the patients were from the country and which were from the city. Oddly, the country boys, with probably limited education and generally using phonetic spelling, offered far more picturesque and informative detail. It did not take much guessing to estimate a man's civilian locale when reading that the noise sounded "like a swarm of bees and locusts around harvest time" or like "a whistle in my ear." The average hearing for conversational tones in the temporal-group tests was 5 ft. on the fracture side and 14 ft. (427 cm.) on the uninjured side. Army normal is considered 15 ft.

Headache, although present in 26 patients, was not generally as disturbing a complaint as the unceasing tinnitus. Of the patients, 50% localized post-traumatic headache to the region of injury, but 25% had no post-traumatic headache. Fourteen per cent described their headache as generalized. It was our impression that generalized headache is a common complaint in battle-weary personnel, and we believe that battle fatigue played a role in this group. All the 13 ears which initially discharged, according to the history, were dry at the time of our examination. There was no evidence of any unresolved septic process in the group. We observed no instances of active labyrinthian irritation. The confusing symptom of "light-headedness" demanded an alertness in the examination of these men for the possible presence of true otologic vertigo, but the patients were seen 9 and 10 months after the injury. Perhaps in some instances, if true vertigo was initially present, time had permitted compensation to ensue. Like generalized headache, "light-headedness" or "giddiness" was a common complaint in battle-weary men.

Longitudinal fractures of the temporal bone are generally associated with a force applied to the side of the head. Bleeding from the middle ear or a spinal-fluid leak is supposed to be a common sequel. In our series there was a history of bleeding or discharging ear after the injury in 12 of the 35 instances of temporal fracture. In some of these cases associated blast injury may have been responsible. Miss D. J. Collier,[19] of the Royal Free Hospital in London, has had a similar experience. She also believes that violence may produce a radiating fracture which cracks the roof of the middle ear, leading to effusion in the tympanic cavity without rupture of the tympanic membrane.

A. Vault Fractures and Neck, Face, Jaw, and Multiple Forms

There were 18 cases in which the site of fracture was the parietal bone and/or posterior portion of the frontal bone. Because of the number of injuries in this

24. Schillinger, R.: Personal communication to the author.

region the group was considered large enough for analysis (Appendix III). Defective hearing was indicated on the questionnaire in every case and in 12 it was bilateral. The average conversational hearing was 10 ft. (305 cm.) in the right and 11 ft. (335 cm.) in the left ear.

In 12 cases the drums were perfectly normal and the conduction mechanism was not especially affected. The lowest incidence of tinnitus was found in this group (61%). We are unable to postulate the reason for this lesser incidence.

Two patients who stated that they had unilateral defective hearing heard conversations at 15 ft. bilaterally; the audiograms at 1024 cps were normal; however, at 2048 cps there was a 40-db. loss.

Three patients who stated that they had bilateral defective hearing showed an abrupt drop from normal at 1024 cps to a 50-db. loss bilaterally at 2048 cps. The remaining nine showed an average loss of 40 db. at 2048 cps, with a normal threshold at 1024 cps. It is evident that if these men were to be screened by the army

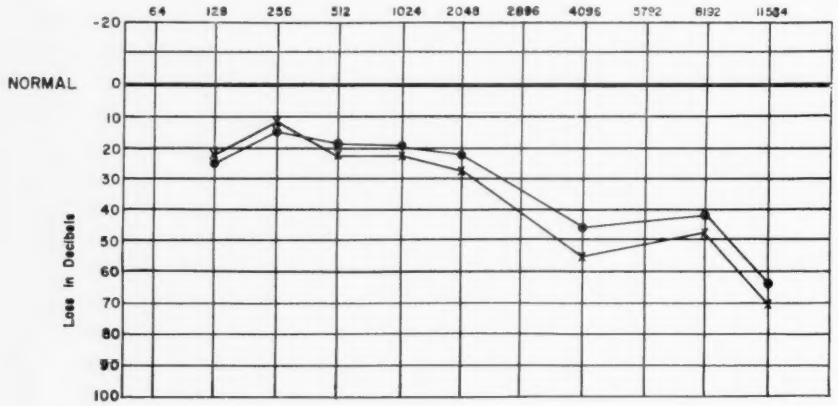

Fig. 8.—Trend audiogram for 18 patients who sustained vault fracture. Air-conduction curves, with dot for right ear and *x* for left.

standard conversational tests they would be acceptable as having normal hearing, but certainly the subjective complaint was corroborated by the abrupt high-tone losses shown on the audiograms, which assured us that the deafness was an honest evaluation by the patients. These high-tone losses (Fig. 8) are significant because of the average young age of these men, who had normal hearing previous to military service. At our examination none of the four patients who had reported otorrhea showed any evidence of it. Debris, dirt, and secondary fragments were the responsible agents for the open dry perforations in four instances, and a blast was responsible in a fifth.

Seven of the vault-fractured patients localized post-traumatic headache. Functional headache is a likely factor in their cases also. It is interesting to note that the 33 patients who had fractures of the face and jaw (Appendix IV) manifested an audiogram trend (Fig. 9) similar to that of the patients with vault fractures. When these two trend audiograms are superimposed they are seen to be identical beyond 2048 cps. As in the temporal-fracture cases, the additional factor of previous exposure to loud noise had some influence on the pattern of the

audiometric curve, although the abrupt drop is more evident here than that of the uninvolved ear in the temporal-fracture series.

B. Concussion Injury of Head

The group of 18 soldiers (Appendix V) who sustained a direct blow without clinical evidence of fracture comprises an interesting series, particularly in the light of experiments done by Stenger.[25] He subjected rats to a blow on the head and found hemorrhage in the first turn of the cochlea and about the round window. Recently, carefully controlled experiments were done by Schuknecht, Neff, and Perlman.[26] They subjected conditioned cats to moderate blows on the exposed skull after surgically destroying one ear. The blow was insufficient to cause fracture. These studies suggested that a blow to the head may create a pressure wave in the skull bones transmitted to the cochlea and resulting in displacement of the basilar membrane without damage to the middle-ear structures. Schuknecht

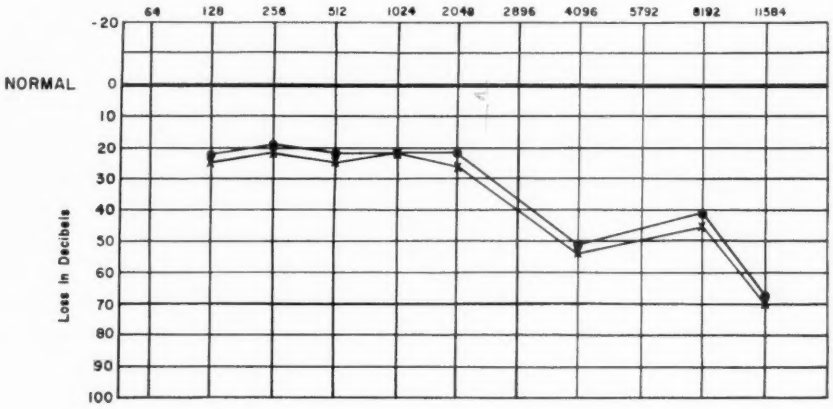

Fig. 9.—Trend audiogram for 33 patients who sustained fractures of neck, face, and jaw and multiple-form fractures. Air-conduction curves, with dot for right ear and *x* for left.

believes that intense stimulation produces a violent displacement of the basilar membrane with injury to the organ of Corti. This situation has also been observed by Davis and co-workers [27] in their experiments on guinea pigs. Schuknecht also found that with moderate blows a mild degree of damage resulted, localized to the 4096 cps level. A new concept in the pathogenesis of this particular localization has been presented. Von Bekesy's [28] experiments with air sound pressures in animals demonstrated displacement of the basilar membrane toward the helico-

25. Stenger, P.: Beitrag zur Kenntnis der nach Kopfverletzungen auftretenden Veränderungen im inneren Ohr, Arch. Ohrenh. **79**:43-67, 1909.

26. Schuknecht, H. F.; Neff, W. D., and Perlman, H. B.: An Experimental Study of Auditory Damage Following Blows to the Head, Ann. Otol. Rhin. & Laryng. **60**:273-289 (June) 1951.

27. Davis, H., and others: Functional and Histologic Changes in the Cochlea of the Guinea Pig, Resulting from Prolonged Stimulation, J. Gen. Psychol. **12**:251-278, 1935.

28. Von Bekesy, G., and Rosenbluth, W.: The Mechanical Properties of the Ear, Handbook of Experimental Psychology, New York, John Wiley & Sons, Inc., 1951, Chap. 27.

trema with low tones and toward the oval window with high tones. He found that at 4000 cps the eddies are free to change their direction of rotation. It has been demonstrated that at this point the basilar membrane is thus structurally deformed, *in situ* so to speak, by the large number of eddies occurring in the spectrum of noises of industry and war, thereby causing damage at the 4096 cps level.

It is striking to note the tonal dip at 4096 cps in our cases (Fig. 10), the same finding being so characteristic in Schuknecht's cat experiments. Naturally,

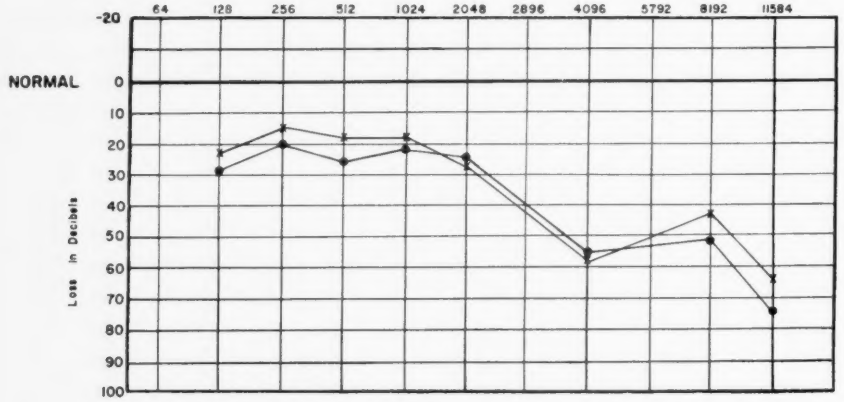

Fig. 10.—Trend audiogram for 18 patients who sustained concussion injury. Air-conduction curves, with dot for right ear and *x* for left.

TABLE 4.—*Decibel Loss of Six Patients Who Sustained Moderately Severe Head Injury*

128		256		512		1024		2048		4096		8192		11584		Conversation Hearing		
R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	Factors in Injury
5	20	5	10	15	20	5	20	5	30	85	65	50	65	45	70	15′	7″	Riding in jeep which struck antitank mine
25	35	10	30	15	30	5	40	5	90	80	85	100	75	100	100	15′	0′	88 mm. shell exploded 15′ to left on D-Day; shrapnel, left vault
10	15	5	5	15	30	15	25	30	35	55	75	35	25	100	70	9′	4′	Lacerated scalp, 30-calibre bullet, Feb., 1944
100	20	100	5	95	5	95	0	95	25	100	60	100	60	100	100	0′	15′	Tank overturned, Sept., 1943
30	50	20	50	30	60	30	55	20	60	70	95	50	80	100	100	2′	0′	Struck by baseball, left mastoid region, May, 1945
20	20	10	10	0	5	0	15	5	45	70	70	30	60	70	100	15′	5′	Lacerated left scalp, 30-calibre bullet, Nov., 1944

the intensity of the blow varied in the human casualties incurred. The average limit of conversational hearing in our cases was 9 ft. (274 cm.) in the right ear and 9½ ft. (290 cm.) in the left ear. This degree of diminished hearing suggests that perhaps more than a moderate blow was sustained by the greater number of these soldiers. Table 4 summarizes the hearing losses of six patients whom we consider to have had moderately severe blows to the head.

The two soldiers who reported discharging ears after injury had additional factors responsible for their hearing defects, and their cases are therefore described.

The patient with bilateral suppuration was a 30-year-old corporal in the Engineer Corps who, while swimming under water at New Guinea on Aug. 18, 1944, was kicked about the left

ear. Otitis media followed. A simple left mastoidectomy was found necessary and was performed Sept. 19. At the time of our examination, March 27, 1945, he could hear at 14 ft. with the dry left ear, and there was a deep, depressed left postaural scar.

The patient who reported left otorrhea was a 24-year-old first lieutenant who was blown out of his tank while in combat on Nov. 18, 1944. He regained consciousness in the hospital, at which time he realized, from the appearance of the bloodstains on the pillow, that both ears had been bleeding. Sand and debris were removed from both external auditory canals. X-rays of the skull were negative for fracture. Our studies, one year after injury, showed a 40-db. loss for conversational tones in the right ear and a 55-db. loss for these tones in the left ear. Vestibular labyrinthian functional studies gave normal results, as did an otolaryngological examination.

It is interesting to compare the trend audiogram (Fig. 10) of the nonfracture head-injury cases with that of the vault-fracture cases. As with the face and jaw fractures, one is impressed with the striking similarity of curve. The existence of this similarity is reasonable, because the only additional factor in the vault cases is evidence of fracture. Actually, our statistics indicate that a blow on the head resulted in a greater degree of hearing defect at the 4000 cps level when there was no clinical evidence of fracture than when fracture was present.

In civilian otologic practice, I have been confronted with judicial decisions as to the cause and effect of head injury on loss of hearing in industrial accident cases. If there is clinical evidence of skull fracture, a claim for deafness compensation is almost automatically allowed. However, with the mere history of a blow on the head without fracture or manifest physical damage to the otologic apparatus, it is not uncommonly disallowed. One can deduce from our unselected cases that there is a high incidence of hearing defect, post-traumatic headache, and persistent tinnitus of significance in patients' complaints in cases without positive physical signs. That these complaints are as generally present in cases without physical signs as in those with such signs suggests that possibly this common legal question needs further consideration.

V. BLAST-WAVE ACOUSTIC TRAUMA

Robinson [29] describes the effect of the detonation of a high explosive as the result of a sudden liberation of large quantities of heat and gas from the casing confining it. The pressure of the gases within the casing has been variously estimated at between 100 and 650 tons a square inch. The heat raises the temperature of the gases to several thousand degrees, and the high temperature produces the high pressure. The pressure blows the casing to bits and the gases burst through with a terrific velocity.

A shock or blast wave is produced by the high-velocity gases as they meet the surrounding medium, air, water, or earth. Since they have great momentum (momentum is the product of mass X velocity), it is transmitted in part to the surrounding medium, and it moves out, also being pushed from behind by the expanding gases.

The word "blast" has been used for this "devastating hurricane" moving at a speed of perhaps 20,000 miles an hour.

There seems to be another effect, the "sympathetic detonation." If the wave is traveling through air or an easily compressible fluid, the increasing pressure in the wave front compresses the gas or fluid behind it and thereby increases its density. This greater density seems to increase the kinetic energy in the advancing wave

29. Robinson, C. S.: Explosions: Their Anatomy and Destructiveness, McGraw-Hill Book Company, Inc., 1944, Chap. 4.

front. The blast wave is a single pulse of increased pressure followed by a wave of suction, and the general form is similar to a single pulse of a large-amplitude sound wave. The maximum pressure of the wave is highest in the region of the explosion, and the pressure falls rapidly the farther the wave moves from the source of the explosion. In the case of a 70-lb. (31.8-kg.) charge, the pressure falls approximately as follows:

14 ft. (427 cm.)	110 lb. (49.9 kg.)
18 ft. (549 cm.)	60 lb. (27.2 kg.)
30 ft. (914 cm.)	15 lb. (6.8 kg.)
50 ft. (1,524 cm.)	6 lb. (2.7 kg.)

These figures represent hydrostatic pressure in excess of normal atmospheric pressure (15 lb. a square inch).[30] Any surface facing an explosion will be subjected not only to this excess pressure but also to pressure due to air movement immediately behind the shock front of the blast wave. Close to an explosion, this pressure may be as great as or greater than the hydrostatic pressure of the shock front, but farther from the bomb the pressure effect of the air is negligible. The slower suction component is always much weaker than the pressure component, and in no case can it be greater than 15 lb. a square inch, since this corresponds to a perfect vacuum. These pressures are enormous if tabulated as acoustic-pressure equivalents.

Perlman[31] analyzed a wave front into its frequency components. A slowly rising front contains a greater portion of its energy in the low frequencies. A sharply rising wave front has a greater portion of its energy in the higher frequencies. He believes that the cochlea acts as a harmonic acoustic analyzer and responds to the pressures accordingly, a steep rise causing greater irritation toward the round window, and a slowly rising pulse causing irritation near the apex. It is of practical importance to note that the advancing wave front of an explosion is uneven, so that objects at distances equally close to the point of detonation may be exposed to various pressures. Farther away from the explosion, the blast wave evens out.

This phenomenon apparently explains what Barrow and Rhoads,[32a] in a study of 200 soldiers exposed to blast, characterize as the capriciousness of blast injury. In the situation they describe, one soldier was resting his chin on the shoulder of another in front of him. The soldier in front was literally "blown to bits," the one behind escaping with a fractured maxilla. They found that the soldiers standing in the front row were seriously injured, while this row of men afforded protection from the blast to the second row, except for the tympanic membrane. There were exceptions, however, for one man standing in the second row, 30 ft. from the explosion, was unconscious for one hour, while his companions were hardly injured. They found that injuries to the eyes and ears were most commonly encountered. Thirty-two men had perforated membrani tympani.

30. Zuckerman, S., and others: Discussion on the Problems of Blast Injuries, Proc. Roy. Soc. Med. **31**:171-192 (Jan.) 1941.

31. Perlman, H. B.: Acoustic Trauma in Man: Clinical and Experimental Studies, Arch. Otolaryng. **34**:429-452 (Sept.) 1941.

32. (*a*) Barrow, D. W., and Rhoads, H. T.: Blast Concussion Injury, J. A. M. A. **125**:900-902 (July 29) 1944. (*b*) Thompson, L.: Shock Waves in Air and Characteristics of Instruments for Their Measurement, J. Acoust. Soc. Am. **12**:198-204 (July) 1940.

Thompson [32b] described the intensity of a shock wave at a distance of 26 ft. (792 cm.) from an explosion of 10 lb. (4.5 kg.) of trinitrotoluene. He computed it as 80 db. above the threshold of feeling, or 200 db. above the threshold of hearing, of periodic sound.

Of our patients, 161 were close enough to an explosion of sufficient charge to subject them to the force of blast. In 50% of the cases heavy-artillery fire was the responsible agent. Many writers, Silcox and Schenck,[16] Marriage,[8] Negus,[33] Cantor,[34] and others, have reported that the inner ear is protected from extensive damage by the dissipation of the force of a blast resulting from rupture of the drum. Wakeley [35] believes that this opinion is exaggerated, and states that rupture of the drum plays no part in determining the absence or the severity of damage to the cochlea. He states that the inner-ear damage occurs before the rupture takes place, and that the severity of the cochlear damage is directly related to the intensity of the blast pressure. To prove this point he describes the experiments of Hallpike and Smith,[36] who demonstrated by oscillograph observations that the reflex contraction of the tensor tympani and stapedius muscles in response to a series of sound stimuli effect a decrease in the amplitude of the electrical response of the cochlea by only 6 db. It is of greater importance that this "damping action" is not developed until 30 msec. after the onset of the stimulus. It is much too late, therefore, to afford any protection against the practically instantaneous blast wave. Following our previous description of the nature of blast wave, we agree with Wakeley that the axis of the external auditory canal in relation to the wave front and the intensity of the blast wave are the two factors which determine whether or not a normal drum will rupture. In connection with "damping effect," it is also interesting to note that Hoshino [37] found that in the case of pistol shots close to the ears of animals in which he had caused suppuration in the middle ear cavity, the attenuating effect of their conduction disability did not protect against detonation injuries to the cochlear labyrinth.

Because of the differences of opinion described, we elected to study separately the blast patients who had ruptured drums. This group comprised 52 patients (Fig. 11). Most ruptures were caused by heavy-artillery fire, with its powerful shock wave (Appendix VI). Ireland's [17] figures are similar to ours. He found that of 317 cases of traumatic blast perforation in World War II, high explosives were the responsible agents in 69.4%, mortar fire in 9.8%, mines in 9.4%, and all other agents in 3% or under. Of the 104 drums in our 52 cases, only 36 were found to be normal nine months after injury. Sixty-six per cent showed

33. Negus, V. E.: Proc. Roy. Soc. Med. **34**:5 (Nov. 1) 1940.

34. Cantor, J. J.: Rupture of Tympanic Membrane Due to Blast: Mechanical Hypothesis, Ann. Otol. Rhin. & Laryng. **54**:554-567 (Sept.) 1945.

35. Wakeley, C. P. G.: Blast Injuries (Dr. John Burns Lecture), Glasgow M. J. **139**:91-102 (April) 1943.

36. Hallpike, C. S., and Smith, A. F. R.: The Origin of the Wever and Bray Phenomenon, J. Physiol. **83**:243-254 (Dec. 31) 1934.

37. Hoshino, T.: An Experimental Study of the Pathology of Ears Injured by Sound and a Consideration of the Physiological Bearing of Results, Kyoto Med. J., 1917; cited by Guild, S. R.: War Deafness and Its Prevention: Report of the Labyrinths of the Animals Used in Testing of Preventive Measures (Middle Ears Previously Reported), J. Lab. & Clin. Med. **4**:153-180, 1918-1919.

at least visual evidence of injury to the drum. We have no way of knowing the incidence of secondary infection after injury, nor do we know about the exact clinical management at the battalion aid station. It is our impression that indiscriminate irrigations were not used, because of the widespread caution to the medical personnel against this ill practice and also because of the demand for immediate attention to the more extensive injuries. Hoople [38] and Hipskind [39]

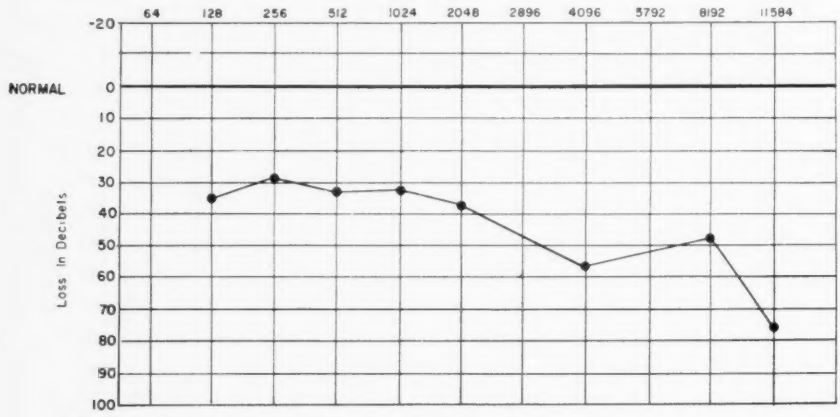

Fig. 11.—Trend audiogram for 52 patients who sustained blast trauma with drum rupture. Of the drums, 36 were normal; 39, perforated; 26, scarred and sclerotic, and 3, retracted. Air-conduction curves.

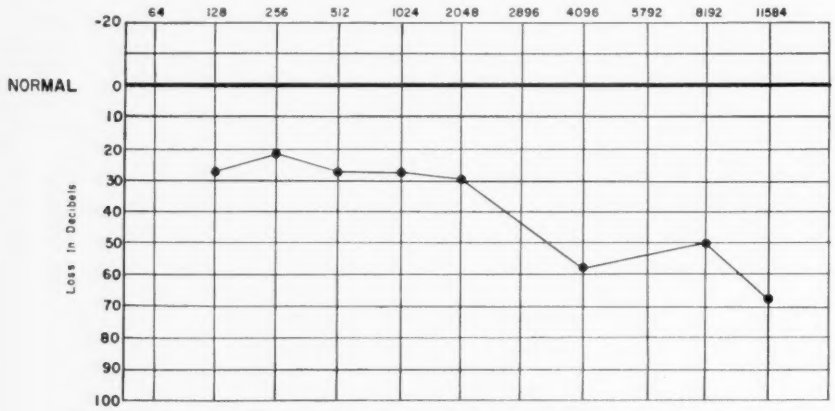

Fig. 12.—Trend audiogram for 182 ears affected by acoustic trauma from blast. Air-conduction curves.

indicated that secondary infection was infrequent in their war experience. Ireland performed simple mastoidectomy in 11 cases of a series of 317. We found no history or clinical evidence of mastoidectomy in any of our 52 drum-rupture cases.

38. Hoople, G. D.: An Otolaryngologist's Experience in World War II, Ann. Otol. Rhin. & Laryng. **55**:121-127 (March) 1946.

39. Hipskind, M. M.: War Injuries to the Auditory Apparatus: Clinical and Audiometric Observations, Laryngoscope 678-705 (Dec.) 1945.

Of the blast patients without drum rupture, 89% complained of defective hearing (Appendix VII); the frequency was 7% greater with drum rupture. This slight difference is perhaps of no more than statistical significance. However, a comparison of the composite audiogram trends for the two groups (Figs. 11 and 12) shows a 10-db. greater drop for conversational tones for the ruptured-drum patients than for the nonruptured-drum patients. From the high incidence of scars and sclerotic healed drums, it is evident that the healing of traumatic perforations is often not a primary union. The resolution of the hemorrhage and the bridging of the gaping irregular perforations, to say nothing of the possible injuries to the ossicular chain and its supporting ligaments, result in permanent fibrosis. Fibrotic changes apparently account for the slightly greater functional impairment of the conducting mechanism as shown by the ruptured-drum trend audiogram as compared with the unruptured-drum trend audiogram. The two show a similar loss for high tones. This fact is significant because it indicates that the rupturing of the drum does not protect the cochlea from blast damage to any great degree. We were not able to obtain statistics as to which quadrant of the drum is most commonly ruptured, but we can conclude from the size and variance of the position of the "effectus" perforations that none of the quadrants are exempt, including the pars flaccida. Two instances were found in which the malleus was conspicuously dislocated from its normal position and another in which the drum was drawn up from below like a wrinkled, raised curtain.

Two hundred and twenty-five patients indicated on the questionnaire that they were exposed to loud explosive noises. They were not in close contact with barrage attacks, nor did they suffer any ear or systemic effect of close blast. In a general way they can be considered as having sustained "fringe"-blast, or peripheral-blast, injury.

This classification was made to determine the acoustic effect of military explosions in the absence of any definite distressing subjective reaction. This group (Appendix VIII) is representative of a very large number of men who were in the front echelons yet not in the midst of direct bombardment. None suffered perforated drums or battle injuries. As in the previous series, the commonest explosive to which they were exposed was artillery fire.

The different thresholds of conversational hearing in this group suggest the variation of susceptibility to acoustic trauma in normal persons, illustrated as follows:

A. 96..... $\frac{15'}{15}$ bilaterally

B. 15..... $\frac{13'}{15}$ to $\frac{14'}{15}$ bilaterally

C. 41..... $\frac{13\text{-}14'}{15}$ one ear; $\frac{0\text{-}8'}{15}$ opposite ear

D. 25..... $\frac{13\text{-}14'}{15}$ one ear; $\frac{9\text{-}12'}{15}$ opposite ear

E. 31..... $\frac{0\text{-}8'}{15}$ bilaterally

F. 10..... $\frac{9\text{-}12'}{15}$ bilaterally

G. 7..... $\frac{0\text{-}8'}{15}$ one ear; $\frac{9\text{-}12'}{15}$ opposite ear

The trend audiograms for subgroups A and E are presented in Figure 13. These subgroups include a little more than one-half of all the men in this group

and indicate the wide variation in susceptibility to acoustic trauma even at a fairly remote distance from the blast wave front. The average duration of service for these men was 27 months; actual combat time unfortunately could not be determined with any degree of accuracy. Hipskind, after studying a small series of blast cases (19), believed that there was no cumulative effect in this type of acoustic trauma. Our studies certainly indicate a persisting defect after an average lapse of 11 months following exposure. Tinnitus was present in 83% of all these cases, apparently associated with acoustic trauma in the 4000 cps range.

VI. PROPHYLAXIS

Headphones were used by some of the personnel firing heavy guns. These served as protection against blast injury to the ears. James,[40] one of many who designed an ear defender in World War II, indicates that even in some stationary batteries these are still not worn. He found that the usual reasons for not wear-

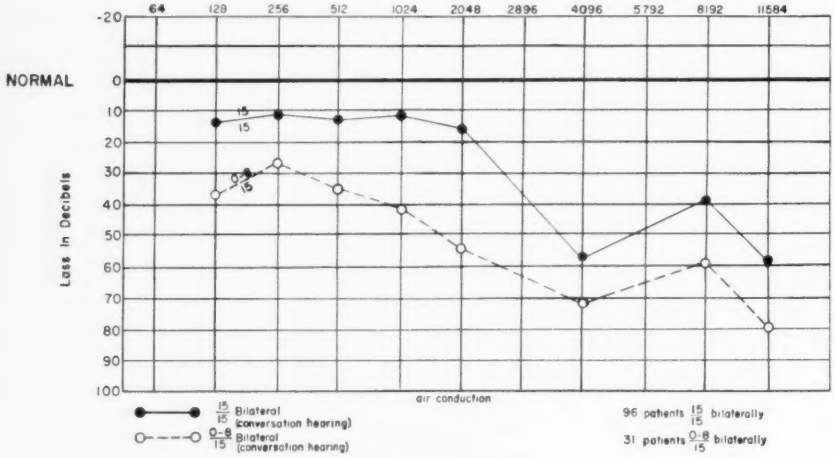

Fig. 13.—Trend audiograms of two groups (Subgroups A and E) of men who sustained "fringe" blast injury.

ing ear defenders were: (1) soldiers thought that they could not hear orders while using defenders; (2) the plugs were uncomfortable and fell out of their ears, and (3) the men could not be "bothered with them."

A number of the men giving the third reason mitigated the effect of the blast by turning the face toward the gun and in this way moving the external auditory canal from the direct line of the muzzle. Others bent the head so that the ear was protected by the brim of the helmet or closed one meatus with the finger and turned that side to the gun while the other hand was busy.

Guild,[10] in 1941, expressed the opinion that ear protectors had been of questionable value up to that time. He did, however, favor the trial of the so-called Tommy protector invented by Trible[41] in World War I. It had provided the

40. James, J. A.: Gun Blast and the Use of Moulded Ear Defenders, J. Laryng. & Otol. **60**:294-297 (July) 1945.

41. Trible, G. B.: Ear Protection, U. S. Nav. M. Bull. **13**:48-60 (Jan.) 1919.

best protection in Guild's animal experiments. It is similar in shape to a medicine-dropper bulb, with a flange collar at the open end to hold it at the conchomeatal junction.

The V-51 Ear Warden is believed by Taylor [42] to be a most effective defender. Ogden [43] found it satisfactory in his testing of various defenders af Laredo Field, in Texas, in 1943. Peyser, Bunch, and Armstrong, according to Larsen,[44] believe that a combination wax and petrolatum tamponade is effective.

Industrious dental officers during World War II reported the construction of custom-made defenders in their dental prosthetic laboratories.[45] Acrylic defenders cast from individual impressions eliminated the external otitis and discomfort of which the men had complained previously, when using ill-fitting defenders made from irritating materials.

In 1950, Schneider [46] stated that suitable defenders reduce acoustic trauma to less than 2%. He found the Rüedi-Furrer model suitable for field service. It consists of headphones with exterior or lateral fenestra and contains within it a Helmholtz resonator with a frequency of 1000 cps to act as a damper. This ear protector does not impede tones up to 800 cps; from 800 to 10000 cps the tones are attenuated between 25 and 30 db., above which loud conversation can be comprehended. Rüedi,[47] in 1952, presented a new defender but admitted that he hoped a more adequate one would be produced.

Peyser,[48] in 1940, devised a test for susceptibility to auditory fatigue. Wilson's [49] test for susceptibility is a refinement based upon Peyser's work. Wilson limited the test to the upper-cycle region of susceptibility rather than considering the whole audiometric scale. The test is based upon the premise that abnormal susceptibility is demonstrated by prolonged recovery time from a measured loud tone. He determines the threshold for 4096 cps for each ear. He then exposes one ear to 2048 cps at 80 db. for a 5-minute period and immediately afterward determines the threshold for 4096 cps at 30-second intervals and then at 8-minute intervals until the threshold is less than 10 db., which is within the limits of error. The opposite ear is tested in the same way. He arrived at his standards by choosing the readings of an intermediate group of tested subjects between low and high values. Recovery in less than two minutes is considered to be normal; three minutes is considered slow, and a duration of fatigue above three minutes is considered definitely abnormal.

Table 5, from Børge Larsen's [44] article on occupational deafness, presents the tests of susceptibility presently used by several otologists. Larsen agrees with

42. Taylor, H. M.: Traumatic Deafness: Problem of Prevention, Laryngoscope **54**:362-373 (July) 1944.

43. Ogden, F. W.: Effect of Gunfire upon Auditory Acuity for Pure Tones and Efficacy of Earplugs as Protectors, Laryngoscope **60**:993-1012 (Oct.) 1950.

44. Larsen, B.: Occupational Deafness, Acta oto-laryng. **41**:139-155, 1952.

45. Cox, L. K., and Geller, J. W.: Anticoncussion Ear Plugs: Prevention of Aural Complications from Gunnery Concussion, U. S. Nav. M. Bull. **43**:139-142 (July) 1944.

46. Schneider, R.: Otological Investigation and Experiments with Ear Defenders on Anti-Aircraft Personnel, Vrtljschr. schweiz. Sanitätsoff. **27**:165, 1950.

47. Rüedi, L.: Occupational Injuries of the Ear, Acta oto-laryng. **41**:118-138, 1952.

48. Peyser, A.: Über die für gewerbeotologische Belastungsprüfungen geeignete Frequenz, Supp. 74, pp. 131-135, 1948.

49. Wilson, W. H.: Determination of Susceptibility to Abnormal Auditory Fatigue, Ann. Otol. Rhin. & Laryng. **59**:399-405 (June) 1950.

Hallowell Davis and his co-workers that a person may vary in his susceptibility from time to time. He also reports that the four tests listed in Table 5 often gave different results for the same person. He sheds doubt on the predictability of the results of these tests, because of Thielgaard's study of 59 weavers with occupational deafness showing a 4096 cps dip. Thielgaard was unable to correlate the size of the dip with the auditory-fatigue effect.

A noise control act has recently been formulated by a committee appointed in 1945 by the government of Finland.[50] It has been presented for legislative action to the ministry for social affairs.

Suggestions as to the prevention of industrial acoustic trauma were recently made by Fox.[51] He believes that industrialists and insurance companies are indifferent to the problem of acoustic trauma. He appeals to otologists to correct this indifference. Aside from the use of ear protectors, Fox recommends the rotation of working personnel and tool redesign to eliminate noise at its source.

War department directives as to prevention of acoustic injury have undoubtedly been beneficial. Yet, despite precautions advised, acoustic trauma persists. The most promising approach to the problem is through the devising of more

TABLE 5.—*Susceptibility to Acoustic Fatigue**

	Peyser	Wilson	Kiml	Thellgaard
	C^3	C^4	C^4	C^3
Fatigue tone	1024	2048	2048	1028
Intensity	100 db.	80 db.	80 db.	100 db.
Duration	3 min.	8 min.	5 min.	5 min.
Interval	15 sec.	2 min.	1 min.	5 min.
	C^3	C^4	C^4	G^3
Effect measured at	1024	4096	4096	1536

* From Børge Larsen's "Occupational Deafness." [44]

efficient ear protectors, which might lessen, but not eliminate, the incidence of acoustic trauma.

VII. SUMMARY AND CONCLUSIONS

A brief résumé of the history of acoustic trauma is presented.

Atresia of the external auditory canal secondary to injury by high-speed missiles was observed. A method of surgical management is described.

A statistical and critical analysis of auditory defects is presented, based upon study of 490 soldiers who sustained head injuries and acoustic trauma.

Of all fractures followed by ear complaints, 40% were penetrating temporal-region fractures. The threshold of hearing on the injured side was elevated to between 40 and 50 db. in the speech range.

Head injuries without fracture resulted in hearing as defective as that due to injury with fracture.

Blast perforations of the membrana tympani do not protect the cochlea from detonation injury.

The 4096 cps tonal dip was characteristic of blast acoustic trauma and more so of head injury.

Forty per cent of the soldiers reporting ear symptoms were exposed to the periphery of explosive blast wave.

50. Sicrala, V.: Noise Legislation, Acta oto-laryng. **41**:139-155, 1952.

51. Fox, M. S.: Acoustic Trauma, Wisconsin M. J. **50**:563-567 (June) 1951.

APPENDIX

I.—*Right Temporal Fracture (Nineteen Patients)*

No. of Patients	Answers to Questionnaire
14 (74%)	Struck by shrapnel
19 (100%)	Defective hearing
	9, bilateral
	10, right
	0, left
17 (89%)	Tinnitus
	11, bilateral
	6, right
	0, left
13 (68%)	Lightheadedness
9 (47%)	Discharging ears (after injury only)
	1, bilateral
	8, right
	0, left
15 (79%)	Headache
	6, temporal right
	4, temporal bilateral
	1, occipital
	1, occipital parietal
	1, frontal
	1, frontal occipital
	1, generalized

Appearance of Drums

12 (63%)	Normal bilaterally
3 (16%)	Healed perforation, right
2 (11%)	Retracted, right
1 (5%)	Perforation, right
1 (5%)	Perforation, left

II.—*Left Temporal Fracture (Sixteen Patients)*

No. of Patients	Answers to Questionnaire
12 (75%)	Struck by shrapnel
15 (94%)	Defective hearing
	2, bilateral
	13, left
	0, right
13 (81%)	Tinnitus
	7, bilateral
	6, left
	0, right
6 (38%)	Lightheadedness (after injury only)
3 (19%)	Discharging ears (after injury only)
	0, bilateral
	2, left
	1, right
11 (69%)	Headache
	7, temporal left
	1, temporal occipital
	1, parietal
	1, frontal
	1, generalized

Appearance of Drums

10 (62%)	Normal bilaterally
3 (19%)	Healed perforation, left
2 (13%)	Retracted
	1, bilateral
	1, left
	0, right
1 (6%)	Sclerotic, left

III.—*Vault Fracture (Eighteen Patients)*

No. of Patients	Answers to Questionnaire
18 (100%)	Struck by shrapnel
18 (100%)	Defective hearing
	12, bilateral
	2, right
	4, left
11 (61%)	Tinnitus
	9, bilateral
	0, right
	2, left
5 (28%)	Otalgia (after injury only)
4 (22%)	Discharging ears (after injury only)
	1, bilateral
	3, right
	0, left
3 (18%)	Lightheadedness
2 (11%)	Dizziness
2 (11%)	Nausea and vomiting
13 (72%)	Headache
	6, generalized
	2, temporal bilateral
	1, temporal right
	1, frontal
	1, frontal right
	1, occipital
	1, parietal occipital

Appearance of Drums

12 (67%)	Normal
2 (11%)	Perforation
	0, bilateral
	1, right
	1, left
2 (11%)	Healed perforation
	0, bilateral
	1, right
	1, left
2 (11%)	Retracted and sclerotic
	2, bilateral
	0, right
	0, left

IV.—*Fracture of Neck or Jaw and Multiple Forms (Thirty-Three Patients)*

No. of Patients	Answers to Questionnaire
29 (88%)	Defective hearing
	15, bilateral
	5, right
	9, left
27 (82%)	Tinnitus
	22, bilateral
	3, right
	2, left
22 (67%)	Lightheadedness
8 (24%)	Discharging ears (after injury only)
	1, bilateral
	2, right
	5, left
24 (73%)	Headache
	7, frontal
	4, generalized
	3, temporal bilateral
	3, temporal left
	2, temporal right
	2, occipital
	1, right temporal and occipital
	1, frontal temporal
	1, frontal parietal

Appearance of Drums

26 (79%)	Normal
4 (12%)	Healed perforation, left
2 (6%)	Retracted
	0, bilateral
	1, right
	1, left
1 (3%)	Perforation, left

V.—*Concussion Injury (Eighteen Patients)*

No. of Patients	Answers to Questionnaire
15 (83%)	Tinnitus
	11, bilateral
	1, right
	3, left
14 (78%)	Defective hearing
	8, bilateral
	2, right
	4, left
2 (11%)	Discharging ears (after injury only)
	1, bilateral
	0, right
	1, left
9 (50%)	Headache
	3, frontal
	2, occipital
	2, parietal
	1, temporal bilateral
	1, generalized

		Appearance of Drums
11	(61%)	Normal
4	(22%)	Retracted and sclerotic
		2, bilateral
		1, right
		1, left
2	(11%)	Perforation
		1, bilateral
		0, right
		1, left
1	(6%)	Sclerotic
		0, bilateral
		1, right
		0, left

VI.—*Blast Trauma Causing Rupture of Drums (Fifty-Two Patients)*

No. of Patients		Type of Explosive
24	(46%)	Heavy artillery
8	(15%)	Land-mine explosion
8	(15%)	Bombing
4	(8%)	Grenades
4	(8%)	Miscellaneous
2	(4%)	Mortars
2	(4%)	Dynamite
		Answers to Questionnaire
50	(96%)	Defective hearing
		30, bilateral
		20, unilateral
43	(83%)	Tinnitus
		30, bilateral
		13, unilateral
38	(73%)	Discharging ears (after injury only)
		8, bilateral
		30, unilateral
20	(38%)	Earache (at time of blast)
		16, bilateral
		4, unilateral
19	(37%)	Lightheadedness (after injury only)
10	(19%)	Nausea and vomiting (after injury)
9	(17%)	Discharging ears (time of examination)
		7, unilateral
		2, bilateral
28	(54%)	Headache
		11, frontal
		7, occipital
		6, generalized
		2, temporal, bilateral
		2, temporal, unilateral

Appearance of Drums

39 (37.5%)	Perforation
36 (34.5%)	Normal
26 (25%)	Scarred and sclerotic
3 (3%)	Retracted

VII.—*Blast Acoustic Trauma (109 Patients)*

No. of Patients	Type of Explosive
60 (55%)	Heavy artillery
18 (17%)	Miscellaneous
14 (12%)	Mortars
8 (7%)	Bombing
4 (4%)	Mine explosion
2 (2%)	Bazooka
1 (1%)	Small arms
1 (1%)	Grenades
1 (1%)	Machine guns
	Answers to Questionnaire
97 (89%)	Defective hearing
	51, bilateral
	46, unilateral
95 (87%)	Tinnitus
	76, bilateral
	19, unilateral
53 (48%)	Lightheadedness (after injury only)
39 (36%)	Earache (at time of injury)
29 (27%)	Discharging ears (after injury only)
20 (19%)	Nausea (after injury only)
76 (70%)	Headache
	15, frontal, bilateral
	4, frontal, unilateral
	4, temporal, bilateral
	14, temporal, unilateral
	13, generalized
	12, occipital
	7, frontal, occipital
	1, occipital, left temporal
	1, occipital, left temporal
	5, parietal

Appearance of Drums

185 (85%)	Normal
26 (12%)	Retracted
	9, bilateral
	8, unilateral
5 (2%)	Sclerotic
	2, bilateral
	1, unilateral
2 (1%)	Scarred

VIII.—*"Fringe" Blast (225 Patients)*

No. of Patients		Type of Explosive
136	(60%)	Heavy artillery
21	(9%)	Mortars
15	(7%)	Miscellaneous
13	(6%)	Bombing
10	(4%)	Machine gun
9	(4%)	Mines
8	(4%)	Dynamite
8	(4%)	Rifle and small arms
5	(2%)	Grenades
		Answers to Questionnaire
189	(83%)	Tinnitus
		149, bilateral
		40, unilateral
165	(73%)	Defective hearing
		105, bilateral
		60, unilateral
68	(30%)	Lightheadedness
55	(24%)	Otalgia
131	(58%)	Headaches
		52, generalized
		35, frontal
		20, temporal unilateral
		12, occipital
		9, temporal bilateral
		3, parietal
		Appearance of Drums
410	(91%)	Normal
30	(7%)	Retracted
10	(2%)	Sclerotic
0		Perforation
0		Scarred

EVALUATION OF DEAFNESS OF NONORGANIC ORIGIN

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WHEN A patient presents himself to a physician, the latter directs his attention to an understanding of the patient as a physiological and psychological entity. Tests, laboratory procedures, and studies of functions are part of an investigation, each being interpreted in terms of the whole patient. A blood cell count, audiogram, or electrocardiogram is no more a portrait of a person than is an x-ray film of the skull. These physical measurements are fragments, each piece contributing its share to the total picture. A discussion of tests utilized in identifying so-called simulated deafness should be considered not as isolated observations but within the context of the whole patient.

Hearing ultimately is a psychological experience. Certain physical environmental changes act as stimuli which alter the minute-by-minute status of the organic physiological receptor acoustic apparatus. The stimulus must be of such dimensions that changes will occur in the receptors. This receptor system is part of a hierarchy in which the psychological mechanism plays a dominant role.

One category of problems with which the otologist is confronted includes patients whose receptor system is impaired, resulting in an amount of defective hearing. Another category consists of patients whose receptors function satisfactorily but whose psychological interpretation of acoustic stimuli becomes distorted, perverted or rejected.

It is this latter group to which we wish to apply ourselves, although the methods of studying the two are basically the same. The patient complaining of a hearing impairment and found to have a serviceable acoustic receptor organ and no cerebral pathology may have developed a psychological rejection to the purpose which the sounds of society obligate him. There are many psychological needs that this rejection may serve, such as withdrawing from responsibility, returning to a status of dependency, escaping from pressing reality. It may also be a method for exploitation deliberately designed for some materialistic gain. Resolving such problems calls upon the resources of the otologist and the psychiatrist, the former to diagnose, clarify, and classify the deafness and the latter to uncover the motivation; one determines the status of the otoacoustic apparatus, and the other elucidates the psychological phenomena.

Priest [1] described his observations among military personnel. He reviewed and evaluated the many tests designed to study unilateral deafness. He emphasized the

1. Priest, R. E.: Tests for Unilateral Deafness, Arch. Otolaryng. **2**:138-143 (Aug.) 1945.

importance of examining the existence of unilateral deafness in military personnel, particularly when the patient denied the deafness, as well as exposing feigned unilateral deafness. He also defined the role of the otologist and that of the psychiatrist.

The tests for identifying deafness of nonorganic origin might be labeled, "tests for the investigation of the complaint of deafness." Be it remembered that deafness is a symptom of disease, whether the pathology is organic or psychologic or both.

The observations begin when the patient first enters the physician's office. The otologist must be on the *qui vive* for clues of behavior, some of which may be gross, others minimal. The value of a patient's history depends not merely on what he says but how he says it, thereby modifying a recital of chronological events of the patient's life by his own interpretations and reactions to these events. One patient may appear overcomposed, another overanxious, another indifferent, another vague, another overexact. Each patient will expose his own behavior trends. Clinical judgment is taxed to the utmost in the effort to evaluate properly such behavior.

The patient's history is explored in great detail; some areas may be reexplored judiciously with different kinds of questions covering the same ground, possibly illuminating inconsistencies in the history. The physician can afford at least a benevolent skepticism of the complaint of any patient until he finds sufficient evidence to substantiate or doubt the patient's own opinions.

To cite some examples: A patient may complain that he cannot hear the telephone bell ring, yet when the otologist's phone rings the patient may promptly stop conversing; or the patient may state that he hears only when he faces the speaker directly, but, if the otologist is careless or clumsy enough to drop an article to the floor, as he stoops to retrieve it he may ask the patient a question which is answered without faltering. Another patient, one who has never had any auditory rehabilitation, may volunteer that he depends primarily on lipreading (self-taught usually); however, the patient carries on and understands a conversation with the otologist while the latter talks with a cigarette between his lips. Thus the possibility that the patient has read the physician's lips is eliminated. The otologist upon completing the history may leave the patient to enter the examining room, calling to the patient to follow. Alacrity in response to such a request may further fortify the physician's suspicions. These are observations of the patient's behavior in an environment with which he is familiar and additive material which may begin to verify or deny his claim to deafness. Hirsch [2] states that the clinical tests of hearing depend on arbitrary conditioning of the moment. The preceding elicit a sample of the patient's conditioning for everyday situations. We think these devices are more sound and reasonable psychologically than those other recommendations so often found in textbooks suggesting that derogatory remarks be made in the patient's presence and his reactions noted or that a coin be dropped to see whether the patient hears the sound of impact.

Generally patients with monaural deafness demonstrate asterophonia, difficulty in localizing the source of the sound, and when addressed from an unanticipated quarter invariably gaze about in order to locate the speaker. The patient with nonorganic monaural deafness generally fails to demonstrate the loss of localization of sound.

2. Hirsch, I. J.: The Measurement of Hearing, New York, McGraw-Hill Book Company, Inc., 1952, pp. 94-95 and 260-275.

Another clue, a very important one, is the patient's speech patterns, as described by Penn.[3] Invariably in the presence of long-standing binaural deafness, abnormalities of speech will appear, the variations depending on the duration of the deafness, its severity, and its type. Of course, factors other than impaired acoustic function will modify one's speech, but development of speech impairment is a concomitant of binaural deafness. Speech of normal intensity, inflection, and quality tends to contradict the allegation of bilateral deafness until adequate explanations may be found. Speech is the consort of hearing, and impairment of the latter often results in deterioration of its companion.

Upon performance of the otorhinolaryngological examination, further clues may be observed. The patient may readily obey instructions given him. During the mirror inspection of the larynx, the patient is asked to close his eyes and then is instructed to phonate so that the movements of the larynx may be noted. The instructions are given at a low conversational level. This examination is often sufficiently distracting that the patient apparently forgets about his hearing, and if his deafness is nonorganic he may obey the instructions given by the otologist at a voice volume which he apparently had been unable to hear during the history interview.

By this time, the otologist will have obtained some impressions to be explored further by the various clinical tests of hearing. Pure tone audiometry is such a test. Often when preliminary observations have suggested that the hearing loss cannot be severe, the air conduction audiogram may demonstrate a most profound loss. There may be marked discrepancies between the audiograms of the two ears; the presumed worse ear may be better audiometrically than the presumed better ear. The audiometric curves may reveal bizarre configurations not ordinarily encountered in organic deafness. The bone conduction thresholds may also demonstrate unusual patterns.

If a continuous frequency audiometer is used, the following sometimes may be observed: Having determined that the loss of hearing appears to be most profound audiometrically, the physician sets the attenuator at about 50 db. for 1000 cps. Then without altering the attenuator, he rotates the frequency dial up to 4000 cps. If the patient signals that he hears the tone as the frequency selector is rotated, thereby changing the frequency only, and then when the tone is interrupted he signals that he no longer hears the tone, there is evidence that he has heard at an intensity which he had previously denied. He has mistaken change of frequency, of pitch to him, for an apparent increase of loudness. This procedure can be repeated at lower intensities. Response of the patient that he has heard changes of frequency which he has interpreted as increases in loudness is evidence that the ear does hear.

If the audiometry is performed by having the patient respond with the word "yes" each time he hears a tone, voice reactions may be observed. The moderately hard-of-hearing person and the normally hearing person usually will react with a change in voice volume to sudden wide fluctuations of intensity of the stimulus from the audiometer. If a patient alleging profound deafness responds with a loud "yes" to a tone of relatively great intensity and immediately with a soft "yes" to

3. Penn, J.: Hearing Defects as Factors Influencing Voice and Speech Patterns, Thesis, New York University, 1952.

the same tone of much less intensity, the physician should infer that at some psychological level the patient has reacted to the changes of the intensities which he has heard and recognized as being of different levels of loudness.

If the patient is observed during the testing through a one-way-vision window, not realizing that he is being watched, he may reveal by his facial and body reactions that he is restraining himself from responding or that he is undergoing an anxiety-producing situation. Similarly other clues may be present. If the patient uses the hand signal to indicate when he hears the tone, he may make small quivering movements of the fingers or hand, as though he is restraining himself from signaling while being aware of the tone. Many hard of hearing patients and normal subjects will demonstrate this kind of hesitating hand motion at threshold. The rate of eye blinking and the respiratory rate may demonstrate changes as though the patient is attentive to the stimulus, although he states that he does not hear the tone. These are additional behavioral manifestations.

Repeating pure tone audiograms at intervals of several days may demonstrate that the thresholds of hearing vary considerably and that these findings are consistently inconstant.

Another test which explores hearing is the speech to noise ratio test described by Doerfler and Stewart.[4] They observed that the normal-hearing ear is able to hear and understand speech at an intensity level less than that of measured masking noise simultaneously introduced with the speech. The truly hard of hearing ear usually will behave in a similar fashion. The patient with nonorganic deafness may state that he cannot hear the words because of the noise or object to a noise level of less intensity than that at which he had apparently been unable to hear speech. This indicates that he has heard less noise (in decibels) interfering with louder speech (in decibels) or he has heard the masking noise at an intensity which he previously had denied.

Another clue during the speech reception test is obtained when the patient responds with half syllables of the spondee words. At other times such patients make up new words in response to the spondee word stimulus. This kind of response except in young children, mentally retarded patients, patients of foreign birth who speak and understand English poorly, and those with severe perceptive deafness should make the observer suspicious of the patient's claims of acoustic impairment.

The Lombard test sometimes exposes binaural alleged deafness and also monaural alleged deafness. Masking noise, such as compressed air from the pressure suction apparatus usually found in a nose and throat office, introduced into both ears while the patient is reading aloud often causes a variety of reactions. If hearing is normal or nearly so, most people will raise their voice volume in response to the masking of their hearing created by the noise in order to hear themselves read. Patients with nonorganic deafness may raise their voice or may read in a halting fashion or mispronounce words. Occasionally the patient will drop the voice to an almost inaudible level. These patients may complain of pain or discomfort or demonstrate some evidence of distress or anxiety.

4. Doerfler, L. G., and Stewart, K. W.: Malingering and Psychogenic Deafness, J. Speech Disorders **11**:181-186 (Sept.) 1946.

The Lombard test helps to confirm or uncover monaural deafness. We perform the test in the following manner: While the patient reads aloud, masking noise is first introduced into the "deaf" ear. If there is an increase in voice volume, we give credence to the thought that the ear does hear. If the voice does not change, we then mask the better ear. If there is an increase in voice volume, we suspect that the "deafened" ear is impaired. If there is no change in voice volume when the good ear is masked, we suspect that the allegedly deafened ear is permitting the patient to monitor his own speech volume. Although we do not rely on the test, we perform it and accept the findings if they confirm other more clear-cut evidence.

The Stenger test is used to evaluate monaural deafness. This test can be performed with tuning forks, as it originally was described, and also with audiometers. We perform the tuning fork test as follows: Using a 500 double vibration fork, we insert the stem into a 90 cm. (36 in.) length of rubber tubing, the diameter of which is about 6 mm. (¼ in.). The other end of the rubber tubing has inserted into it an olive tip such as is used with auscultation diagnostic tubes. A blindfold covers the patient's eyes. The patient is instructed to raise his hand and hold it up only as long as he hears the sound in the good ear. The olive tip is inserted into the auditory meatus of the patient's good ear and remains so inserted throughout the test. When the fork is vibrated, the subject will hear the tone in the ear bearing the olive tip. The tone arrives at the ear from the fork through the lumen of the 90-cm. tube. Then the tines are brought close (about 3 in. [7.6 cm.]) to the opposite "deaf" ear. The binaural normally hearing subject will signal that he no longer hears the tone in the first ear when so tested. The patient with significant organic monaural deafness will signal that the good ear continues to hear the tone. However when the tines are brought up to the "deaf" ear of the patient with monaural nonorganic deafness, he will signal that the good ear no longer hears the tone. His reply is that of a binaural normally hearing person.

The Stenger audiometry test requires more elaborate equipment, producing more precise results. One audiometer and two receivers with an additional switch and circuit before the phones can be used, or two balanced audiometers, each with its own receiver, or a two-channel audiometer are equally serviceable for the test.

Usually the frequencies of 500, 1000, and 2000 cps. are tested. The better ear will receive the tone at an intensity level of about 15 db. greater than its threshold. The patient reports that he hears the tone in the good ear. The same tone is then poured into the impaired ear at a level of 15 db. greater than that which the good ear is hearing. A patient with normal hearing will report that the "good" ear, or the one which first heard the sound, no longer hears it. When the tone to the second, or "bad," ear is interrupted, the patient reports that he hears the tone in the first ear again. Actually, the two ears receive the same tone simultaneously although at two different intensities, the greater intensity in the second, or "bad," ear masking, cerebrally, the tone in the first, or better, ear receiving the lower intensity. Patients with organic monaural deafness will not succumb to this masking until the intensity in the deaf ear is relatively large. Patients with nonorganic monaural deafness will demonstrate the masking phenomenon. Two conclusions may then be drawn: First, the alleged deafened ear does hear, and, second, we know that the threshold for the particular frequency is no higher than the number of decibels which masked the

normal, or better, ear. For example, if 15 db. in the good normal ear is masked by 30 db. in the "deaf" ear, then the threshold of the "deaf" ear for a given frequency can be no higher than 30 db. and may be lower (better). Testing the masking thresholds of the middle three frequencies and determining that a positive Stenger test is obtained at approximately serviceable hearing levels for these frequencies is evidence of at most minimal acoustic loss for the ear so tested.

The testing of hearing by pure-tone audiometry with a psychogalvanometer [5] offers one method whereby the cooperation of the patient by volitional signaling can be dispensed with. Reasonably accurate audiometric pure-tone thresholds can be elicited usually if technical, physical or mechanical factors do not interpose artifacts or uncontrollable variables. When reliable positive results are obtained, they can be given as much credence as subjective tests. Interestingly, many patients realizing that their hearing has been tested "electrically" without any contribution on their part, as by signaling, often give reasonably accurate subjective audiometric thresholds immediately thereafter.

The apparatus consists of an audiometer, galvanometer, and faradic inductorium. Audiometric headphones are placed on the ears; one pair of electrodes leads from two fingers of the patient to the galvanometer, and another pair leads from the inductorium to the patient's arm. To perform the testing the observer exposes an audiometric tone to the patient at an intensity which he believes that the patient hears, or at which the patient admits hearing. After the tone, the patient receives an electric stimulus from the inductorium. The uncomfortable experiences, such as that of a mild electric shock, produce a change in skin potential. The change of skin potential is registered by the galvanometer. Repetitions of the cycle of tone-shock condition the patient so that eventually there will develop skin potential changes galvanometrically observable when the tone alone is heard. When this occurs, the tone is exposed to the patient at decreasing intensities until the galvanometer indicates no change subsequent to the stimulus of the tone. The last audiometric intensity reading at which the galvanometer responded is the presumed threshold of hearing for the tone so tested. Repeated testing in this manner will determine whether valid results are being obtained. The auditory spectrum of each ear then can be explored in an orderly manner.

Recently Lee [6] described his observations of artificial stuttering. He performed the following experiment: The subject, while wearing headphones, reads printed material into a microphone. The reading is recorded on tape and played back to the subject through the phones at a delay of anywhere from 0.04 to 0.28 second. Hence the subject hears via the phones what he has just read a fraction of a second after he has spoken it. This played-back speech invokes disturbances in further reading, resulting in stuttering as well as other vocal or verbal manifestations. This observation suggests that with appropriate calibrated equipment one might be able to measure organic binaural deafness of equal magnitude binaurally as well as expose binaural nonorganic deafness.

5. Bordley, J. E., and Hardy, W. G.: A Study in Objective Audiometry with the Use of a Psychogalvanometric Response, Ann. Otol. Rhin. & Laryng. **58**:751-760 (Sept.) 1949.

6. Lee, V. S.: Artificial Stutter, J. Speech & Hearing Disorders **16**:53-55 (March) 1951.

CONCLUSIONS

The symptom of deafness of nonorganic origin, monaural or binaural, is observed not infrequently in clinical experience.

Tests of hearing can substantiate the patient's complaint of deafness.

These same tests of hearing can demonstrate that the alleged deafness is not the result of significant oticorganic pathology.

Hearing tests cannot expose the motivation for the complaint of deafness, discovered to be of nonorganic etiology.

The motivation must be uncovered by psychiatric investigation.

Psychogalvanic objective audiometry is feasible and, frequently, clinically reliable.

The study of the entire patient, his history, his behavior, his speech, his physical status, and the adequate testing of auditory function will be productive of competent conclusions.

115 E. 61st St. (21).

RECRUITMENT TESTING IN HEARING AND ITS IMPLICATIONS

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AUDIOLOGY is becoming an increasingly important diagnostic science and as such may provide important clues to what are now considered to be rather obscure problems. It is our intention to present some of the audiologic techniques now in use for testing for the presence of recruitment in hearing and to evaluate these tests in terms of their validity and availability. At this time, it might be well to review some of what has been written in this area of interest.

The literature on recruitment presents a rather confused picture at the present time, but this paper will attempt to describe some of the methods now in use.

Recruitment of loudness is a phenomenon involving a foreshortening of the usable range of hearing. A subject with recruitment exhibits an abnormally rapid acceleration of loudness units for a given range of physical intensity units. In other words, though hearing of a recruiting ear and a normal ear might, at threshold, be a considerable distance apart, once one passes threshold and increases the intensity of a signal, a convergence of loudness sensation is evident between the two ears. The recruiting ear starts late but catches up to the other ear in the intensities above threshold.

Chart 1 illustrates some of the different types of acceleration that have been described.

TECHNIQUES FOR DISCOVERING RECRUITMENT

Probably because of the relatively recent heightened interest in recruitment testing there are currently in use a rather large number of different techniques for discovering it in an ear. The existing methods may roughly be divided into the following two categories: Loudness balance testing and difference limen testing.

A. *Loudness Balance Testing.*—This technique employs the more direct approach of the two, in that the acceleration of loudness in an affected ear, or at an affected frequency, is compared directly with that in a normal ear or at a frequency taken as normal. A direct comparison between two loudness accelerations is the distinguishing feature of the method. There are various modifications of this technique.

1. Fowler's Binaural Method:[1] The acceleration of loudness in an affected ear is compared directly, on the basis of the patient's report, with that in the normal ear

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1. Fowler, E. P.: Diagnosis of Diseases of Neural Mechanism of Hearing by Aid of Sounds Well Above Threshold, Laryngoscope **47**:289-300 (Aug.) 1937.

of the same patient. This method is obviously limited in application, since it requires the patient to have a normal ear for comparison. It is generally accepted, however, as the most valid test of recruitment for experimental purposes, inasmuch as the data are direct. Its chief limitations are in its usefulness and the complexity of its administration.

2. Fowler's Monaural Method: This is a refinement of Fowler's binaural method to extend the method's applicability. Monaural deafness is not required for this technique. The loudness acceleration at a frequency at which recruitment is suspected is compared with that at some other frequency in the same ear, taken as normal. Even with this refinement, however, there are still many patients who cannot be accurately tested with the method. It requires that some frequency in an

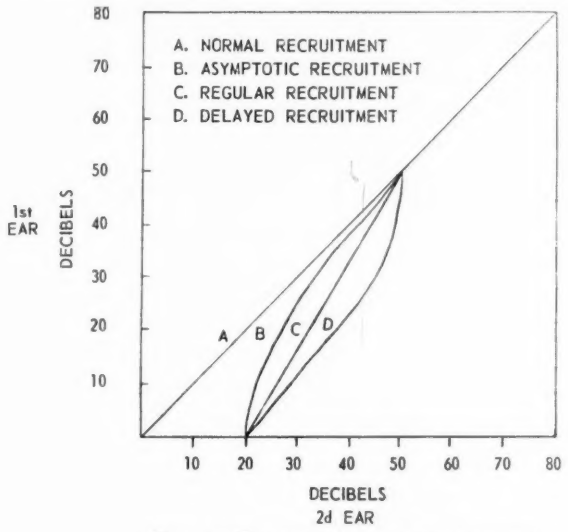

Chart 1.—Three recruitment increment curves.

affected ear behave normally on loudness acceleration, in order to serve as a criterion. If there are no such frequencies, the method is useless.

3. De Bruine-Altes' Masking Method:[2] This method is not essentially different from Fowler's original technique. It seems to be an attempt to reduce the subjectivity of the patient's comparisons by measuring the masking effect of a frequency at several intensities by introducing a tone of lower frequency simultaneously with it. The chief objection to this technique is that it requires even more elaborate equipment and testing procedures than does the technique of Fowler. It is also subject to the criticisms of range of applicability.

4. The Range of Loudness Method:[3] It is surprising to find so little attention given to this technique in the literature. A reasonably careful survey turned up only one reference to it.

2. De Bruine-Altes, J. C.: The Symptom of Regression in Different Kinds of Deafness, Thesis, University of Groningen, Holland, 1946.

3. Watson, L. A., and Tolan, T.: Hearing Tests and Hearing Instruments, Baltimore, Williams & Wilkins Company, 1949, pp. 86-87.

One of its most commendable features is its simplicity. Any audiologist can learn the technique in a few minutes, and the only equipment required is any standard audiometer. No special attachments or gadgets are necessary.

The method involves the determination of two suprathreshold loudness levels, one called the "most comfortable loudness level" and the other called the "uncomfortable loudness level." The advantages of this technique, beyond its simplicity, are that:

(*a*) It can be used on most subjects in a comparatively short time. We were able to test five frequencies in each ear in about 15 minutes, on the average.

(*b*) Inasmuch as it gives a picture of loudness acceleration in each ear at several frequencies, it can be used as a binaural and monaural method combined.

(*c*) Inasmuch as a criterion is set up which remains, as far as possible, the same from subject to subject, it may be possible to standardize the method so that an "absolute" measure of recruitment can be perfected.

(*d*) If recruitment exists, since the technique gives two separate levels, a fair estimate can be made as to whether it is "early" or "delayed" recruitment. We believe that this information may eventually become diagnostically important.

The disadvantages of the method are few:

(*a*) There is a possibility that criteria may change. It is obvious to any clinician who has ever used this method that the criterion of "comfort" or "discomfort" may change in a subject during the testing procedure. However, this change usually occurs only as an adjustment in one direction or another, and it seems to settle down after the first judgment or two has been made.

(*b*) There may be a difference in criteria between individuals. Many persons with normal hearing prefer to listen to sounds which are comparatively faint, while others prefer their sounds comparatively loud. The uncomfortable loudness level is generally less variable than the most comfortable loudness level in this respect. This situation would result in variability between individuals as to the most comfortable loudness measure and consequently in a reduction in validity of this measure as an absolute test of recruitment. Part of the purpose of this study was to determine the normal limits within which the most comfortable and the uncomfortable loudness levels might be expected to vary. It may be pointed out here, however, that even if the measure should not be accepted as an absolute one, the technique would still be a promising one to provide a comparative measure, like the loudness balance techniques previously discussed.

(*c*) Probably the most serious limitation of the technique is that in cases in which the threshold loss is severe and recruitment is small, it is impossible to find the uncomfortable loudness level with standard audiometric equipment. In such cases, the most comfortable level remains, unless, of course, the loss is so severe that even this possibility is excluded.

B. *Difference Limen Testing.*—The rationale behind all the tests in this category is that, if abnormally fast loudness acceleration (recruitment) takes place, then the loudness units (the difference limina) must be smaller, when compared with a scale of physical intensity, than would be the case for a normal ear. Chart 2 represents the reasoning behind difference limen testing.

If the distance, in terms of physical units of stimulus, between the threshold and any point above threshold in a recruiting ear is smaller than the distance between threshold and the same loudness level in a normal ear, and if the number of loudness units, of difference limina, remains the same, then the difference limina must be smaller than in the normal ear. (Of course, it might be argued that it is theoretically possible for the number of difference limina to be reduced while their size remains the same, so that a grosser difference discrimination is present than in the normal ear. Though theoretically possible, this situation does not seem to have occurred in any recognized case of recruitment in the literature.[4])

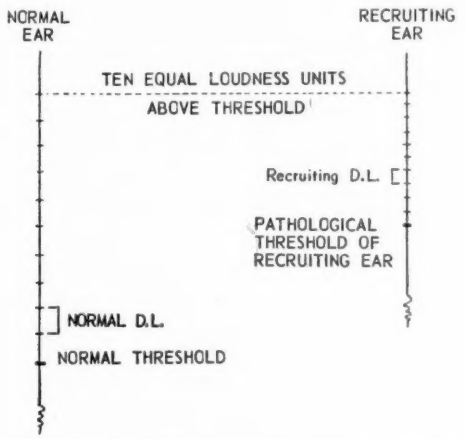

Chart 2.—Rationale of difference limen testing for recruitment.

The difference limen tests are all based on the conclusion that the difference limen in a recruiting ear will be reduced. Some of the techniques of difference limen testing are as follows:

1. Bekesy Method:[5] This method requires a special audiometer which presents to the subject a tone constantly varying in intensity. It can be set so that the frequency also varies constantly, from low to high, if it is desired to check the entire range of hearing rather than a given frequency. The subject, in effect, records his own audiogram, varying the intensity output from just barely audible to just barely inaudible. This procedure supposedly gives the size of the difference limen at threshold.

The advantages of this technique are obvious:

(*a*) It can be carried out easily, being fully automatic except for the instructions. It is also a quick test.

4. We have recently made tests in one case in which the difference between threshold and uncomfortable loudness level in one ear was very small (less than 5 db.), while the threshold difference limen as measured by the Bekesy audiometer was fairly large (about 7 db.). However, there may have been a communication problem operating. If not, this is the only case we have come across in which this possibility may have clinical substantiation.

5. Bekesy, G. V.: A New Audiometer, Acta oto-laryng. **35**:411-422, 1947.

(*b*) It can be used to yield a threshold audiogram at the same time that it gives the difference limen size at threshold.

(*c*) It can be used to measure a continuous frequency range or to measure any discrete frequency, as the operator chooses.

The disadvantages are serious:

(*a*) It is expensive.

(*b*) The results are sometimes difficult to quantify.

(*c*) Individual uncontrolled factors can greatly affect the results (factors such as reaction time, the amount of sound needed to make a decision by the subject, the attention factor, and the sense of rhythm).

(*d*) According to the reports of most of our subjects, it is tiring and unsettling.

(*e*) The most usual technique of obtaining measurements only at threshold entirely misses any delayed recruitment. If other measurements are obtained, the time of administration is increased, thus detracting from one of the advantages of the method.

2. The Lüscher-Zwislocki Method [6]: This method involves the alternate presentation of two tones, identical in frequency but varying in intensity, at about 40 db. above threshold. (Lüscher states that this level above threshold may be reduced to 15 or 20 db. without fundamentally affecting the results.) The difference between the tones is gradually lessened, and the subject is required to signal when he no longer hears the tone variations. The difference between the tones (in percentage of pressure) is then recorded and used as a measure of difference limen size.

The advantages of this technique seem to be that:

(*a*) It is a fairly quick test.

(*b*) It is fairly inexpensive. An attachment to create the desired effect can be built for about $75.00 and can be hooked up after slight modification to a standard audiometer.

(*c*) It gives an absolute measure of recruitment, so that differences between ears, or even between frequencies in a given ear, need not be present.

Some disadvantages that may be mentioned are that:

(*a*) It does not appear altogether reliable. It has been the experience of several investigators that sometimes normal persons will exhibit a difference limen smaller than the critical range for recruitment, especially with practice, and frequently known recruiters will exhibit a normal-sized difference limen.

(*b*) Jerger [7] has pointed out that a technique using a descending difference between the two tones is likely to cause a sort of after-pulsation to persist in the patient's awareness and mentions that some of his subjects reported a variation when none existed. We might point out parenthetically here that we occasionally found the same situation in using the ascending method. After a couple of presenta-

6. Lüscher, E.: The Difference Limen of Intensity Variations of Pure Tones and Its Diagnostic Significance, Proc. Roy. Soc. Med. **43**:1116-1128 (Dec.) 1950.

7. Jerger, J. F.: A Difference Limen Recruitment Test and Its Functional Significance, Laryngoscope **62**:1316-1332 (Dec.) 1952.

tions, some subjects reported a difference before one was ever introduced. We believe great care should be taken in difference limen testing to avoid this reaction.

(*c*) Denes and Naunton [8] have criticized this technique on the basis that it may be measuring a qualitative difference limen rather than a quantitative one.

(*d*) Reaction time and the amount of variation needed for decision by the patient can also influence this test. For example, we heard rather frequently from subjects that they would "wait until the fourth pulsation," or thereabouts, before signaling, in order to be sure.

(*e*) If the difference limen is taken at only one intensity level at a given frequency, an assumption is made that all recruitment is a fairly regular process throughout the intensity range at that frequency. Several authors have reported differing increment patterns for different subjects.

3. The Denes-Naunton Method: This is essentially a refinement of the Lüscher-Zwislocki method. The pulsation effect is eliminated by asking the subject to say which of two sounds is louder. The sounds are presented separately rather than in the continuously alternating method of Lüscher. Denes and Naunton begin with two tones which are considerably separated in intensity. Then the tones are gradually brought closer together until the subject reports them as equally loud.

The difference limen is measured with this technique at 4 db. and at 44 db. above threshold. The indication of recruitment is the difference between the difference limen size at the two intensity levels. If the difference limen increases from 4 db. to 44 db. or remains unchanged, recruitment is said to be present. If it decreases, recruitment is absent.

This method has the advantages of difference limen testing in general, plus the following:

(*a*) It yields a comparative measure between two intensities. If a subject required an unusual amount of information to make a decision, that factor should apply to both the 4 db. level and the 44 db. level. Using the difference between the two levels controls a good number of otherwise uncontrolled factors.

(*b*) It eliminates the pulsation "after-image."

(*c*) It covers a fairly large sample of the intensity range at a given frequency.

The disadvantages are that:

(*a*) If the technique is used with a standard audiometer, in which the intensity steps are graduated in units of 5 db. each, it is difficult to be sure exactly where a subject's threshold is. For example, if he responds at 35 db. and not a 30 db., then his threshold may be 31, 32, 33, 34, or 35 db. If one puts in a tone 5 db. above his "threshold" at this frequency (above 35 db., that is), the presented tone may be as little as 5 db. or as much as 9 db. above the threshold. A difference of as much as 5 db. at a level so close to threshold can be of great importance in estimating the size of the subject's difference limen.

(*b*) This method assumes that recruitment is always of the "early" type, beginning no later than 4 db. above threshold. It further assumes that recruitment is of

8. Denes, P., and Naunton, R. F.: The Clinical Detection of Auditory Recruitment, J. Laryng. & Otol. **64**:375-398 (July) 1950.

the kind which Harris and Myers [9] call "asymptotic," that is, that recruitment is steepest at threshold and gradually curves into the same cort of loudness acceleration which one gets in a normal ear.

Thus, the method would reveal asymptotic recruitment (Chart 1, Line *B*) but would not reveal regular recruitment, Line *C*, or delayed recruitment, Line *D*. As a matter of fact, in cases of delayed recruitment, which does not begin until after 4 db. above threshold and is still acting strongly at 44 db., the Denes-Naunton method would present exceptionally clear-cut evidence against recruitment.

(*c*) There are two difficulties encountered in obtaining the 44 db. level difference limen. Frequently deafness is so severe that the ordinary audiometer is not capable of putting out 44 db. above the subject's pathological threshold. Also of importance is the fact that frequently recruitment is so sharp that the subject's uncomfortable loudness level is reached long before one gets 44 db. above threshold, and consequently the subject cannot bear to listen to the sound long enough for the examiner to obtain a difference limen for him at this level.

4. The Jerger Method: Jerger has refined the Lüscher method and improved on it in the following ways:

(*a*) He uses the ascending method (beginning with no difference and gradually increasing the difference until the subject hears a pulsation).

(*b*) He uses 15 db. above threshold as his level of presentation. This is a much more practical clinical level to test. Jerger apparently agrees with Doerfler, to whom he refers, in the statement that the greatest differentiation between a normal and a recruiting ear is to be found between 10 and 30 db. above threshold.

The disadvantages of this method are the same as those of Lüscher's technique, with the exceptions mentioned.

The present study attempts a comparison of four of these methods.

PROCEDURE [10]

A. *Range of Loudness Method.*—This loudness balance technique was chosen because it is the one most available to the average clinician and needs only a standard pure-tone audiometer for its execution. Threshold, most comfortable loudness level, and uncomfortable loudness level were obtained for each subject, somewhat after the fashion described by Watson and Tolan. After threshold air-conduction audiograms were obtained for both ears subjects were told:

> I shall present the tone very faintly and gradually increase its loudness. When the tone reaches the point at which it is most comfortable for you to listen, not too loud and not too soft, press the signal key. It is that point at which you might listen to a radio for a long period of time. What we want is the most comfortable level of loudness for you.

The tone was given at least three times at each frequency. If the subject's responses fell within 5 db. of each other, the level which was chosen twice was taken

9. Harris, J. D., and Myers, C. K.: Loudness Perception for Pure Tones and for Speech, Manual of Medical Department, Navy, Medicine & Surgery Bureau, Medical Research Laboratory Report 156, Bu Med Project, NM 003 041. 21.05, P 97-127, 1950.

10. All tests were made with the subject seated in a sound-treated room with an ambient sound level of less than 24 db. as measured by a General Radio sound-level meter, Type 1551 A. set on Scale A. The examiner sat with the testing equipment in an adjacent control room and viewed the subject through a small window.

as the measure. If more than 5 db. of variability was obtained, the tone was repeated enough times for the subject to give three responses with a spread of no more than 5 db. among them.

The frequencies 1000, 3000, 500, 2000, and 4000 were tested, in that order. Then the 1000 cps frequency was repeated, to see whether the subject's criterion had changed during the test. If the new measure fell within 5 db. of the former measure, the operator proceeded to the next step. If the new measure deviated by more than 5 db., the new measure was recorded for 1000 cps and a recheck was done at 3000 cps, and so on, until stability was achieved.

The test for uncomfortable loudness level was conducted in exactly the same manner, except that the instructions were now:

The next test is very similar to the last one. This time, however, wait until the tone becomes uncomfortably loud. As soon as the tone becomes so loud that it is distinctly uncomfortable, press the key.

The results were charted on regular audiogram blanks.

B. *The Bekesy Test.*—This was done with a Bekesy-type of continuously variable audiometer, as described by Reger.[11] The intensity control was set for a 2 db. increment second, and the frequency control was set to traverse five octaves in five minutes, from 250 cps to 8000 cps.

Quantitative measurements were something of a problem. The most obvious method is direct measurement of the distance from the point of the "peak" to the point of the "trough." However, this method would involve a great deal of careful measurement with finely graduated units for any workable measure. Also, since the amount of recruitment at the discrete frequencies of 500, 1000, 2000, 3000, and 4000 cps was needed for comparison with the other techniques, it would have been difficult to decide which distance (trough to peak) to use, since there is some variation between the adjacent measures in this respect.

Inasmuch as the number of changes of direction, or peaks, in a given frequency range is a function of the length of upward and downward strokes of the pen, it was decided simply to count these points in a given frequency range and use this number as our measure. The range decided upon was one octave, with its midpoint at 500, 1000, 2000, 3000, and 4000 cps. This range should be large enough to smooth out incidental differences, but small enough to be fairly discriminative.

C and D. *The Lüscher-Zwislocki and the Denes-Naunton Method.*—In this study, the size of the difference limen at 45 db. above threshold was used for the Lüscher-Zwislocki method rather than that at 40 db. in order to combine this method with the Denes-Naunton method. The difference between taking the difference limen at 40 db. and taking it at 45 db. should not be great enough to influence the results significantly. The difference limen at 5 db. and at 45 db. was taken at 1000, 3000, 500, 2000, and 4000 cps, in that order. The ascending method was employed in an attempt to avoid any after-pulsations.

The subjects were given the following instructions:

You will hear a smooth, steady tone over the earphones, like this. After you listen to the tone for a while, it will break up and begin to pulsate, like this. (The subject is given a sample of

11. Reger, S. N.: A Clinical and Research Version of the Bekesy Audiometer, Laryngoscope **62**:1333-1351 (Dec.) 1952.

no pulsation and maximum pulsation at the appropriate times.) You have just heard the smooth tone and the extreme of pulsation. Somewhere between these extremes is a point at which you will first hear it begin to pulsate. When that happens, press the signal key.

The instructions were amplified if the subject did not seem to understand.

The tone was then presented to the subject at 1000 cps at 5 db. above his obtained threshold. The intensity difference was slowly increased until the subject signaled that he heard the pulsation. Pulsations were then immediately discontinued and the difference in db., to the nearest 0.25 db., was recorded. This procedure was carried out five times for each of the five frequencies used in this study, and the median measure was taken as the difference limen size.

The simplicity of the difference limen apparatus used in these observations should be emphasized. Containing only one pair of tubes in a simple switching circuit, the unit constitutes merely an accessory used in conjunction with an ordinary audiometer.

The pair of tubes—in this case gas-filled triodes—is operated in a free-running switching circuit, the two tubes having approximately equal "on" and "off" times. In the plate circuit of one tube is connected a sensitive relay, the normally open contacts of which are used intermittently to short circuit an adjustable resistance at a rate of approximately 40 times a minute. By appropriate insertion of this dropping resistance in series with the plate supply of the oscillator stage of the audiometer, there result periods of reduced output level interspersed with equal periods of normal output level. Varying the magnitude of the resistance, which may be calibrated for direct reading, makes possible observation of the difference limen, in the present apparatus over the range of 0.1 to 5 db.

The procedure was repeated at 45 db. above threshold when a reading was obtainable. For many of the deafened ears, this procedure was not possible, and readings were therefore obtained at maximum output of the audiometer, and all measures which represented a difference between the lower and louder measure of 20 db. or more were used. Lüscher believes that there should not be any significant difference between the difference limen at 40 db. and that at 20 db. Apparently Denes and Naunton believe that for recruiting ears the difference limen is no larger at threshold than at any point up to 40 db. Normal ears, in their reckoning, should show at least 3 db. difference between difference limina in a 20 db. spread, so that it should be permissible to use the measures as described.

The results of this testing procedure were used in two ways:

1. The absolute sizes of the difference limen at 5 and at 45 db. were checked for their value as absolute measures, after Lüscher.

2. The difference between the difference limen at 5 db. and that at 45 db. was investigated to check the efficiency of the Denes-Naunton method.

SUBJECTS

There were two groups of subjects used, a normal group and a deafened group. The normal group consisted of 13 women and 4 men, or 34 ears. There were 2 subjects between 18 and 19 years of age, 11 who were 20 through 29 years old, and 4 who were between 30 and 39. No subject was used who showed more than a 10 db. loss at any single frequency.

RESULTS

Inasmuch as no independent criterion was available, the technique chosen for investigating the validity of the four tests of recruitment was really a technique for discriminating. It was felt that, since each measure used in the study is supposedly a measure of recruitment and nothing else, and could not therefore be influenced by any difference between the normal group and the deafened group other than a difference in recruitment, a simple technique of studying the discriminating efficiency of the tests between the normal group and the deafened group should yield a fairly good indication of validity. Consequently, the difference between means was computed for each measure at each frequency, and the sigma of each difference was found. Then *t*-ratios were arrived at by dividing the difference between means by the sigma of the difference.

The Denes-Naunton technique (difference limen, 5 db. and 45 db.) differentiated between the normal and deafened groups only at 4000 cps. The difference limen at 45 db. differentiated at the 0.01 level of confidence only at 2000 cps and at 4000 cps. The difference limen at 5 db. above threshold differentiated at the 0.01 level of confidence only at 2000 cps and 4000 cps. The Bekesy technique, the most comfortable loudness minus threshold technique, the uncomfortable loudness level minus most comfortable loudness, and the uncomfortable loudness level minus threshold all differentiated at the 0.01 level for all frequencies tested.

By far the most discriminating technique was the range of comfortable loudness method, with the uncomfortable loudness to threshold measure easily the best of the three measures in this method. It should be mentioned here that the results are somewhat spurious, in that a necessary selection in favor of discrimination between the groups was working. In cases of severe threshold loss, ears with marked recruitment would be more likely to be testable with the standard audiometric equipment used in this study than would those with little or no recruitment. Thus, the selection of cases would swing the data to some extent. We can only say that our clinical experience indicates that such spuriousness is not extreme and that the measure of uncomfortable loudness to threshold appears in practice to be considerably more valid than either of the other two very valid measures derived from the range of comfortable loudness test. The Bekesy seems to be the next most valid technique, followed by the difference limen at 5 db. above threshold, the difference limen at 45 db. above threshold (Lüscher's general technique), and the difference limen at 5 db. minus the difference limen at 45 db. (the general Denes-Naunton technique).

SUMMARY AND CONCLUSIONS

Let us quickly review the necessary characteristics of a good recruitment test.

1. It must be as valid as possible, that is, it must measure recruitment. If we can assume, as mentioned, that the only difference between the normal group and the deafened group, at least as far as the measures used are concerned, is a difference of recruitment, then it appears that both the range of comfortable loudness technique and the Bekesy method are valid beyond the 0.01 level of confidence, with the exception of 500 cps on the uncomfortable to most comfortable loudness measure. This is explainable on the basis that recruitment occurs in the higher frequencies much oftener than in the lower frequencies and should be expected to do so in our deafened group. The difference limen testing, in general, does not seem to dis-

criminate as well as the range of comfortable loudness and the Bekesy techniques, and the Denes-Naunton method seems to do so least of all. Harris and Myers [9] have suggested that the asymptotic type of recruitment, which is the only type for which this technique is designed, may actually be in the minority. On the basis of validity, then, it appears that the Bekesy method and the range of comfortable loudness method are both acceptable, with the latter considerably better than the former.

2. A recruitment test should be applicable to all types of hearing losses. Most loudness balance techniques fail in this respect, requiring either a monaural loss or at least one normal frequency in the ear tested in order to set up a criterion of normalcy. The range of comfortable loudness method, however, appears to be acceptable as both a relative and an absolute measure for recruitment. It also appears to measure all types of recruitment, early or delayed, asymptotic or straight-line. The Bekesy method and the difference liment test at the 5 db. level demand early recruitment for their efficient functioning. The Denes-Naunton technique requires not only early recruitment, but asymptotic recruitment. The Lüscher method requires the presence of recruitment preferably at 40 db. above threshold (and early recruitment might be entirely missed because of this) or at least 15 or 20 db. above threshold. The various distances other than 40 db. have not yet been fully investigated.

The chief objection to the range of comfortable loudness technique on this matter of applicability is that the uncomfortable loudness to threshold measure is occasionally not obtainable. However, this objection is not too serious, for two reasons. First, if recruitment does exist to a marked degree, it will frequently show up on the uncomfortable loudness to threshold measure even if the subject has a severe loss. Second, if this measure cannot be obtained, there is always the most comfortable loudness to threshold measure to fall back on. The same difficulty, depending upon the limits of the equipment used, applies to the Lüscher and Denes-Naunton methods.

3. A recruitment test should be inexpensive. It is important to the audiologist or the otologist that recruitment tests be within the scope of his budget. The cheapest method discussed in this study is the range of comfortable loudness technique, inasmuch as there is no equipment necessary other than a standard audiometer. Next in cost comes the difference limen tests other than the Bekesy, all of which can be done with the device mentioned. The most expensive of the group is the Bekesy.

4. A recruitment test should, as a secondary consideration, be simple to administer and to interpret. The range of comfortable loudness and the Bekesy, we believe, should rank about equally as to this requirement, with the Bekesy having a considerable edge in ease of administration and the range of comfortable loudness having the edge in interpretation.

In view of these considerations, it appears that the most desirable technique for measuring recruitment is the range of comfortable loudness technique.

CHANGING CONCEPTS IN OTOLARYNGOLOGY

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A SURVEY of our group of otolaryngologists reveals a senior group of members, at least in part foreign trained, and a junior group, trained within the United States. I propose to make a few comments on the training and outlook of these two groups.

Training for our specialty has been standardized, minimum requirements have been established in the basic sciences, and a proper balance has been proposed for training programs in the clinics and in the hospitals. The plan was largely pioneered by those who were instrumental in founding or who served as members of our first examining board. Witness the names of George E. Shambaugh Sr., Harris P. Mosher, Joseph C. Beck, Thomas E. Carmody, Thomas H. Halsted, R. Clyde Lynch, Hanau A. Loeb, Burt R. Shurly, Ross H. Skillern, Frank R. Spencer, and William P. Wherry. Of this group only the kindly Dean of Otolaryngology, Harris P. Mosher, survives, in Marblehead, Mass., to observe the developments of the program.

Consider for a moment the dilemma of those who attempted training in our specialty before this plan was formulated. There were, here and there, excellent preceptorships for the occasionally fortunate trainees. These did not provide training in the basic sciences.

The importance of the basic science approach was realized by many groups similar to ours. In our own group J. Gordon Wilson, formerly a teacher of anatomy at the University of Chicago, and George E. Shambaugh Sr. carried on gross and microscopic studies in the anatomy laboratory, supplemented by pathologic material from various hospitals. Many hundreds of slides were prepared serially from many temporal bones, and the reports of the important findings greatly enriched the growing knowledge of the fundamental pathology of otosclerosis. The later use of antibiotics made possible the surgical correction safely accomplished in modern fenestration surgery.

Joseph C. Beck and Frank Novak Jr. stressed the necessity of greater knowledge of gross pathology and histopathology. They urged the contribution of important specimens to the Army Medical Museum Armed Forces Institute of Pathology and stressed the value of an atlas of pathology of otolaryngology. The new atlas, soon to appear, has 2,000 illustrations and provides a new, 100-slide study set. It is designed for otolaryngologists and pathologists. To quote Dr. E. P. Fowler Jr.

President's address read before the Chicago Laryngological and Otological Society, May 4 1953.

"The new atlas compiled by Dr. Ash and Dr. Muriel Raum will be part of the personal library of every good general pathologist in the world and from its illustrations will come the lantern slides which will edify the medical students and head-and-neck surgeons for years to come."

Arthur Proetz, an honorary member of our society since 1931, and his St. Louis group studied the physiology of the nose and paranasal sinuses. The impact of these studies on otorhinolaryngology was extraordinary and their contributions invaluable. These are but a few of the examples of fine basic work that formed the groundwork for the basic science course.

When members of our senior group started their training, they could not find within the United States planned teaching programs adequate in scope. This led in some instances to total training in clinics abroad but usually to supplemental training chiefly in London, Berlin, and Vienna.

It is interesting at this time to note that the London Eye and Ear Infirmary was founded in 1805; the New York Eye and Ear Infirmary ear section, in 1822; the Philadelphia Institute for Diseases of the Eye and Ear, in 1822; the Massachusetts Eye and Ear Infirmary, in 1824; the Baltimore Eye and Ear Institute, in 1840, and the Illinois Eye and Ear Infirmary, in 1858. It is important to recognize that these hospitals were first staffed by general surgeons especially interested in eye and ear diseases. Growing interest produced the American Ophthalmological Society in 1864 and the American Otological Society in 1868. Interest in specialization developed through the balance of the nineteenth century, but the establishment of eye and ear hospitals ceased because general hospitals began to include all specialties. The American Board of Ophthalmology was established in 1917 largely through the efforts of William H. Wilder, and the American Board of Otolaryngology in 1924, largely through the efforts of George E. Shambaugh Sr. These were the first specialty boards, and they soon became a stanch challenge to those who assumed the role of specialists without adequate preparation.

Nowhere were there better teaching facilities, better clinical material or a broader scope of graduate training than was offered in Vienna at the Vienna General Hospital and its affiliated teaching hospitals associated with the University of Vienna. The American Medical Association of Vienna, founded in 1904, developed interest slowly at first, was dormant during the First World War, but was reactivated in 1921. The importance of this training center is attested to by the fact that from 1879 to 1940 over 20,000 English-speaking physicians and surgeons attended postgraduate courses. In the seventeen years from 1921 to 1938 alone, 11,800 specialists and trainees from English-speaking countries throughout the world, but chiefly from the United States, attended University of Vienna courses. At the height of activity 75% of the students came from the United States, 25% from the British Isles, Canada, Australia, Egypt, South Africa, India, Palestine, China, Japan, the Phillipines, Hawaii, and Brazil. A recent review of the courses given and of the men who gave them still commands deep respect. The clinics in otorhinolaryngology were headed by Neumann, Hajek, and Alexander and were beautifully reequipped after the First World War by the Rockefeller Foundation. Popular courses were given by Oscar Hirsch, who had many instructive specimens on which to demonstrate variables in anatomy and pathology. Erick Ruttin gave a clinical approach to ear diseases and their complications to capacity classes. Victor Fruhwald taught Halle's endonasal sinus surgery and plastic surgery with ample cadaver

and clinical material. Froschels and Stern taught the pathology and therapy of speech and were then using laryngeal stroboscopy. Franz Haslinger taught and demonstrated clinical laryngology, intubation, tracheotomy, and bronchoesophagology. Many other qualified teachers did excellent work with unlimited cadaver and clinical material. The graduate student could obtain authoritative knowledge promptly and spend most of his time correcting his known deficiencies.

Shortly before returning from Vienna, I assisted Gustave Alexander with a radical mastoid operation performed under local anesthesia, using sterile cotton gloves. Rubber was scarce. After the operation was completed, I went with him to his private office. In his mail for the morning was a small sleek-looking package from George E. Shambaugh Sr. He looked at me while partially opening the package and said, "You are from Chicago; do you know Dr. Shambaugh?" I replied, "I do; he helped me arrange my training program." He then said, "The package contains some slides on otosclerosis. The Americans have fine paper and packaging," and he put the slides on a shelf. Soon after returning to Chicago, I visited Dr. Shambaugh Sr. and he informed me that he had recently sent some unusually excellent otosclerosis slides to Gustave Alexander. I stated that I was present when they arived. He asked. "What did he say?" I replied, he said, "The Americans have fine paper and packaging," and put the slides on a shelf. Then Dr. Shambaugh Sr. answered, "What is it the Bible says about casting pearls to the swine?" The incident was amusing, but I am certain that in proper time those slides were critically studied and courteously acknowledged. Gustave Alexander had many of the gentlemanly qualities of the late Dr. George DeSchweinitz, who so greatly strengthened the Department of Ophthalmology at the University of Pennsylvania.

There were other training opportunities, usually narrower in scope and with more language difficulties, in Germany, Hungary, England, France, and Spain. The Germans, still stung by defeat in the War in the early 1920's, were generally less friendly.

Hitler terminated training opportunities in Vienna in 1938. This date marks the beginning of two important new developments. First, otolaryngology, always a highly surgical specialty, began at this point to undergo a transition with the advent of antibiotics. Those unconvinced stated that antibiotics were dangerous because they would mask symptoms. Acute mastoid surgery, intracranial complications of ear and sinus infection, and deep neck infection requiring surgery were to become more and more infrequent. There has come to be a new conception of what constitutes surgical limits and emphasis in the practice of otolaryngology. Second, we in the United States, recovering from a serious postwar adjustment, began seriously to provide adequate training facilities within our borders to meet the transition challenge. Many of you have had a part in formulating this program.

Several facts now present themselves:

Our membership has not decreased. It has increased.

You cannot train young men to do acute mastoid surgery that pediatricians prevent. You can train young men to do reconstructive temporal bone surgery for deafness; indeed, most residents in otolaryngology include Chicago or New York training in fenestration techniques. With such training ample provision is made for the occasional acute surgical mastoiditis and the chronic diseases originating in the middle ear requiring surgical care.

You cannot attract trainees to the field, after time-consuming and expensive training, and then deny them for many months of the year tonsil and adenoid surgery on account of fear of poliomyelitis. The answer to this is not immediately evident. We hope that it will be through the early development of an effective antivirus for poliomyelitis.

Our population is increasing. Our trainees are not. We may encounter a period of lush practice for properly trained otolaryngologists who can give normal routine care in the specialty as well as the skillful handling of technical otolaryngological problems.

The senior age group in our population is increasing. This will require more and more study and training in the effective care of patients with malignant diseases. This will include many new irradiation techniques, better neck dissections, and better general and special geriatric care.

Patients with frequent colds and definite allergy manifestations often limited to the respiratory tract, giving symptoms for which we are consulted, offer a phase of medical otolaryngology. If these patients are referred to a general laboratory or to a general allergist for testing, they will probably not achieve an ideal therapeutic result. If you will learn to give, or have given under your direct supervision, the simple key tests, chiefly inhalants, and associate the reaction with known clinical responses, you can learn to give controlled hyposensitizing therapy that will render a very valuable service to a frequently poorly treated group of patients.

As long as people drive cars and as long as foreign alliances keep us continually at war, there must be training in head and neck, plastic, and maxillofacial surgery, all of which should be in our field. There is, of course, also a legitimate scientific field for cosmetic surgery.

Certain persons with unusual capabilities and opportunities can profitably practice in large centers of population exclusively otology, rhinology or laryngobronchoesophagology.

Audiological studies are necessary, but exhaustive ones are time-consuming and frequently are carried out by nonmedical personnel. While this offers assistance for case-finding for fenestration surgery and for cross filing of hearing defects for large clinics, we should not let it become to otology what optometry has become to ophthalmology.

We have within our ranks many who have national and international prominence. To give a list would run the risk of failing to give proper credit in some instance. We commend those of you who through unusual effort and ability have won this recognition.

The field of psychosomatic medicine, especially in times of stress, should not be neglected in otolaryngological practice, nor should central nervous system disturbances, metabolic disturbances, nutrition, biochemistry, and water balance studies. Otolaryngologists must be constantly a part of medicine in general.

Our otolaryngology section courses of the American Academy of Ophthalmology and Otolaryngology must be geared to the newest concepts in the practice of otolaryngology, and new candidates for training in otolaryngology have to be shown an adequate return for their time, effort, and expense in training.

Each generation produces its great physicians, but no other age has been able to provide such fabulous opportunities for medical advancement. Atomic medicine

is a new frontier. The susceptibility of the vestibular system and of other vulnerable parts to release of radiation energy from radioactive materials used in therapy or irradiation from atom-powered motors poses new problems. High altitude and space medicine have involved otology in at least two ways: aerotitis and acoustic trauma.

Colonel Paul Campbell, of Randolph Field, Texas, has given a few challenging facts. Rockets operate at maximum efficiency in the space above the atmosphere, carry their own oxygen and air-conditioning equipment, and operate independently of the atmosphere. Complete anoxia occurs at 52,000 feet, and at 66,000 feet body fluids boil if the cabin is not pressurized. The skin temperature of a V2 rocket measured 647 C. at one stage of its penetration of the atmosphere. One might well include an asbestos suit for the beginning and end of the trip, and if a sealed cabin, which is required, is punctured by a high-velocity meteoric fragment, it would literally make the occupant's blood boil. Above 12 miles, the ozone is at a toxic level and cosmic rays constitute an as yet insurmountable barrier.

Coming back to earth we find that 62 diseases of man, from common colds to poliomyelitis, are of viral origin. Progress has been made possible in studying them by (1) discovery of methods of propagating and purifying them, (2) perfection of the electron microscope, and (3) development of basic knowledge of cell physiology and biochemistry, since viruses are obligate intracellular parasites. This group is highly important, since all viral infections, except those caused by large viruses of the lymphogranuloma-psittacosis group, have failed to respond to any known antibiotics. It is not improbable that viruses, occurring naturally or produced by artificial mutation, which will be therapeutically effective against certain types of tumors, may soon be discovered.

Changing concepts are not limited to otolaryngology. I believe that this is a great age in which to practice medicine. Better medicine is being practiced. We anticipate many phenomenal discoveries. Otolaryngology will continue to be a strong specialty with some changes. Our society will contribute much to the scientific progress of otolaryngology.

VACCINATION FOR CHRONIC SINUSITIS AND CHRONIC NASAL CATARRH

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IN A PREVIOUS communication [1] we published the results of our work on the following subjects: (1) chronic sinus disease and chronic nasal catarrh; (2) cough and expectoration still present after a partially successful pneumothorax treatment or thoracoplastic operation; (3) pulmonary tuberculosis with troublesome cough and sputum for which no surgical procedure could be employed; (4) bronchiectasis, and (5) allergic bronchitis.

In the present communication, we are presenting our work to date with chronic sinusitis and chronic nasal catarrh. Patients with chronic nasal catarrh showed no evidence either by x-ray examination or by transillumination of sinus disease.

METHOD OF STUDY

Preliminary Investigations.—Before beginning treatment and as a routine measure, we make a thorough physical and x-ray examination of each patient. X-rays of all the sinuses are made; transillumination of the sinuses is also done, and a careful physical examination of the entire body is made. Urine, sputum, and blood are thoroughly studied. With each patient, an exact study of all the organisms found in the sinuses and posterior nares is made and carefully recorded. This has been done since 1928 and will be reported in this communication. The importance of a thorough study of the lungs by both x-ray and physical examinations was shown in several of our cases in which the constant drainage of discharge from the sinuses down into the lungs during sleep caused serious and permanent disease in the lung structure, or bronchiectasis.

Preparation of Filtrates and Autogenous Vaccines.—Materials for culture are taken with absorbent cotton swabs from both posterior nares and from the sinuses when necessary. The assistance of a laryngologist is often advisable. The discharge is immediately streaked on blood agar plates and on Sabouraud's agar and is also cultured in dextrose broth and in thioglycollate broth for anaerobes.

At the end of 24 or 48 hours the cultures are examined. The organisms are then subcultured for identification. Usually all of the types of the organisms are used in preparing the vaccine and filtrate. The following procedure is employed.

1. A pure culture of each organism is grown separately in a dextrose broth flask for 48 hours at 37 C.

2. The broth culture is centrifuged for 30 minutes at 3,000 rpm.

3. The supernatant fluids of all cultures are saved and combined for the preparation of sterile filtrate.

1. Kinghorn, H. M.; Wilson, G. E., and Dworski, M.: Bacterial Filtrates and Autogenous Vaccines in the Treatment of Chronic Sinus Disease and Other Chronic Respiratory Affections: Preliminary Report, New York J. Med. **49**:41 (Jan. 1) 1949.

4. Bacterial sediment of each strain of organism is suspended in salt solution (0.85%) and poured into individual test tubes containing glass beads. The tubes are sealed and shaken by a machine until an even suspension is obtained.

5. The shaken suspension of each strain of organism is filtered through a cotton filter to remove any clumps.

6. The number of organisms present in each tube is counted in a Hauser-Petroff counting chamber. Usually the suspension is diluted, so that each cubic milliliter contains 40,000,000,000 organisms. From this concentrated suspension further dilutions are made. Dilutions of the various organisms are now mixed and placed in vaccine bottles, the first bottle containing a combined total of 2,000,000,000 organisms per cubic centimeter. The second bottle contains 20,000,000,000, and the third bottle, 40,000,000,000 of the mixed organisms.

7. The bottles are sealed and heated at 56 C. for one hour.

8. Sterility tests are made by adding 0.1 cc. of each mixed vaccine to thioglycollate broth and incubating for 48 hours at 37 C.

9. Phenol is added to make a 0.5% solution.

Filtrate Solutions.—1. The supernatant fluids of all cultures are mixed and are sterilized by passing through a Seitz bacterial filter.

2. Sterility tests are made by incubating the filtered broths for 72 hours.

3. If the broth is sterile, phenol is added to make a 0.5% solution as a preservative.

Determination of Sensitiveness.—To determine whether the patient is sensitive to the vaccine and filtrate, 0.05 cc. of vaccine dilution containing 2,000,000,000 organisms per cubic centimeter, equivalent to 100,000,000 organisms per cubic centimeter, and 0.05 cc. of filtrate are injected intracutaneously into the forearms—vaccine into the right arm and filtrate into the left arm. The results are observed and recorded at 5-, 10-, and 15-minute intervals and at the end of 24 hours. Two types of reactions may occur: Within 15 minutes, an immediate reaction may appear, characterized by a wheal, often 10 mm. in diameter, surrounded by an erea of erythema, often 30 to 40 mm. or more in diameter. This reaction is similar to the one produced in persons sensitive to horse serum or white of egg. Measurements of the wheal and of the erythema are recorded. These reactions may fade within a few hours and may be followed by the second type of reaction, which resembles a tuberculin reaction, producing induration and erythema from 2 to 5 cm. in diameter. Sometimes one reaction occurs without the other, but either type seems to indicate general sensitiveness. If no skin reaction results, the patient is probably not sensitive to the vaccine and filtrate, and treatment with this vaccine and filtrate will likely be of no value.

METHOD OF TREATMENT

It is advisable to avoid general reactions in the administration of the vaccines and filtrates. We therefore inject them intracutaneously into the forearms—vaccine into one arm and filtrate into the other—and with each successive dose alternate from one arm to the other. We believe that a better vaccination is obtained by the intracutaneous method. Constitutional reactions are also avoided. Marked and extensive hyperemia usually follows the injections, especially with the filtrate solutions. The hyperemia from the filtrate solutions occurs almost immediately. The injections are begun with small doses, 0.05 cc. each of vaccine and filtrate, and are given once a week. The same doses of vaccine and filtrate are given three times; then the amounts of both are increased by 0.05 cc.—the dose always being repeated three times—until 1 cc. each of both vaccine and filtrate is injected.

The first dose of 0.05 cc. of the vaccine solution should contain about 100,000,000 organisms. When the amount of injection of vaccine reaches 1 ml., the second bottle, containing 20,000,000,000 organisms per milliliter, is used. Dosage with this second bottle starts with 0.1 cc. and increases as with the first bottle.

We use a tuberculin syringe, Betz No. 226, to give the injections. Syringe and needles should be boiled before use. If a very extensive hyperemia results, the dose should be repeated or reduced. If fever results, the dose should be reduced to two or three previous lower doses and then increased as before.

Treatments can continue four to six months. Improvement or cure may not be permanent. Then another course of injections may be given. Sometimes there may be no apparent improvement, but later, when treatment has ceased, improvement or even cure may occur. If the patient complains of headache, pain in the back, and/or indefinite pains and aches following a dose, even though there is no fever, the dose should be either reduced or repeated. When the dose is reduced, the reduction should be to about three doses. It can then be increased, as mentioned above, by 0.05 cc. amounts.

TABLE 1.—*Results of Treatment in Sinus Disease*

Case	Organisms Isolated	Immediate Results	End-Results, 1952
R. J. C.	Proteus vulgaris, Staph. aureus, hemolytic Streptococcus	Immensely improved	9 yr., good, no relapse
E. G.	Staph. albus	Cured	18 yr., cured, no relapse
F. L. S.	Staph. aureus	Immensely improved	3 yr., good, no relapse
C. H. G.	Hemolytic Staph. aureus and nonhemolytic Streptococcus	Immensely improved	3 yr., good, no relapse
L. W.	Staph. aureus	Cured	11 yr., cured
G. M.	Hemolytic Staph. aureus	Cured	10 yr., cured
M. McC.	Hemolytic Staph. aureus	Cured	10 yr., partial relapse
E. C. McC.	Hemolytic Staph. aureus	Cured	Relapse
I. C. C.	Hemolytic Staph. aureus and nonhemolytic Streptococcus	Immensely improved	11 yr., no relapse
W. McC.	Staph. aureus	Immensely improved	9 yr., relapse
A. D. C.	Hemolytic Staphylococcus and nonhemolytic Streptococcus	Not improved	Not improved
R. F. L.	Hemolytic Staph. aureus	Immensely improved	7 yr., partial relapse
M. T. S.	Staph. aureus and Staph. albus	Cured	6 yr., cured
P. E. C.	Staph. aureus and Staph. albus	Not improved	6 yr., greatly improved
M. McA.	Hemolytic Staph. albus	Improved	Relapse
G. W. A.	Nonhemolytic Staph. aureus	Immensely improved	Relapse

As already mentioned, patients are given their autogenous polyvalent vaccines and filtrates intradermally. Also, filtrates are put directly into the sinuses when necessary and are always applied directly by absorbent cotton swabs into the middle nares in the following manner: The interior of each nasal cavity is sprayed with 10% cocaine solution. The filtrate solution is applied to both nostrils by introducing fully saturated cotton packs to the middle meatus in the region of the hiatus semilunaris, where one finds the openings of the antrum, the anterior ethmoid cells, and the frontal sinus. Small packs are also applied to the inferior and middle meatus. When we wish to put the filtrate solution into the antrum we introduce it either through the natural opening of the antrum in the middle meatus or puncture under the inferior turbinate into the antrum. Selection of method depends upon the nature of the case. In some persons it is very difficult to enter the antrum by the natural route. In either method, however, we cocainize the region (under the anterior end of the middle turbinate or under the inferior turbinate) with 10% cocaine solution. About 2 cc. of filtrate solution is put into each antrum. The treatments are given weekly. Each treatment is followed by the usual nasal oil sprays.

The patients treated with the vaccines and filtrates were divided into two groups —one, those with true sinus diseases verified by x-ray, and the other, those with chronic nasal catarrh. In Table 1 the organisms isolated and the results of treatment are listed. A Staphylococcus was found in all instances in both groups of patients.

This study required the cooperation of an internist, a laryngologist, and a bacteriologist. The internist looked after the physical condition of the patient and gave the injections into the arms; the laryngologist made the injections into the sinuses and the nose and the bacteriologist prepared the vaccines. Treatment was thus not difficult. Most of the patients were ambulatory. The number of injections depended upon the response to treatment. In some patients there was no apparent improvement. Later, however, improvement or even cure sometimes occurred. Patients who showed no improvement were classified as "unimproved."

The intradermal injections and the treatment for the sinuses were usually done on the same day.

We are giving the case histories of all of our patients, since they offer valuable information and round out our study.

REPORT OF CASES

Sinus Disease

R. J. C., a man aged 47, an office clerk, consulted us in June, 1943. His sinus disease began when he was 18 years of age and had thus lasted 29 years. There was a very offensive discharge from the nose, mostly from the left nostril. When he would bow his head, discharge would run from the nose. He used from six to eight handkerchiefs a day. Cultures from the left antrum showed a Gram-negative bacillus, a Staphylococcus aureus, and a hemolytic Streptococcus. The Gram-negative bacillus was identified as a Proteus vulgaris. Vaccine and filtrate solutions were prepared from all three organisms. The patient was treated with the vaccine and filtrate as usual, and the filtrate solution was injected into the left antrum and put into the middle nares on swabs. The left antrum and the left frontal sinuses were involved. Both antra had permanent openings, and the left one was draining steadily. The result of treatment was exceedingly satisfactory. The patient had no head colds the following winter although he had always suffered from winter head colds. The odor from the nasal discharge disappeared, and some days he did not need to use a handkerchief. He became immensely improved, almost cured, and has remained well. His treatment lasted three months, with visits once a week.

E. G., a trained nurse, aged 26, was seen by us in July, 1934. In 1931, an abscessed molar tooth had been extracted, and her sinus disease began a short time later. It became so troublesome that she had to go to Arizona for the winter months. On x-ray examination, the disease seemed to be confined to the left antrum and left frontal sinus. Cultures from the left antrum showed a Staphylococcus albus. Vaccine and filtrate were made. The vaccine and filtrate were given intradermically, and the filtrate was put into the left antrum, about 5 minims (0.3 cc.) at each treatment. The patient became completely cured, and all discharge from the nose ceased. She has remained in New York State since treatment. If a head cold ever develops, it lasts only the usual time, then ceases entirely, and does not affect the sinuses. She has remained cured. Her treatment lasted five months, with visits once a week.

F. L. S., a civil engineer, aged 48, was seen by us in September, 1939. He had suffered from asthma since October, 1919. At that time his nose was filled with polyps. These were burned, and thereafter the asthma developed. Polyps were removed from time to time with the snare. Sinus trouble had been present for about 16 years. When seen by us, he complained of coughing in the morning until he almost gagged, and asthma, mostly at night, which lasted all through the year. He raised a clear mucus about eight times a day and used eight handkerchiefs a day for his nasal catarrh.

Materials for culture were obtained from the right and left antra and the frontal sinuses and gave a pure culture of Staph. aureus. Vaccine and filtrate were prepared and given as usual, and the filtrate was put into the nose on swabs.

His right lung showed emphysema at the base, diaphragmatic adhesion, and probably a slight thickening of the pleura in the apex. His left lung showed emphysema at the base, somewhat exaggerated paravertebral linear markings, and thickening of the pleura in the apex.

X-ray examination of the sinuses showed clouding of both antra and probably of the frontal sinuses, with definite thickening of the turbinates.

The decrease in discharge from the nose reduced the use of handkerchiefs from eight to two a day. The amount of sputum from the lungs diminished remarkably. The asthma, however, continued, and the patient died of its effects some years later. His treatment lasted two months, with visits twice a week, at three to four day intervals.

Mrs. C. H. G., a trained nurse, aged 62, consulted us in February, 1940 on account of severe cough and profuse expectoration. At the age of 18 she had begun to have prolonged winter coughs. These occurred for a number of years and would cease only in the summer. In 1934, she again had severe winter cough with expectoration, and this condition continued till the present time. She had had a nasal catarrh for years, although the catarrh would occasionally cease entirely for a considerable time. When seen by us, the nasal discharge was very profuse, and she coughed and raised sputum. The cough occurred mostly in the morning and during the night and was usually accompanied by expectoration. For several years she had a slight wheeze in the chest.

X-ray examination showed the linear markings throughout both lungs, more particularly those to the bases, to be somewhat exaggerated, suggesting a chronic bronchitis. The heart shadow was somewhat enlarged in the region of the left auricle, an appearance which would suggest the possibility of a heart lesion.

Roentgenograms of the cranial sinuses showed definite and rather pronounced clouding of the left antrum. Cultures from the posterior nares yielded a hemolytic Staph. aureus and a nonhemolytic Streptococcus. Autogenous polyvalent vaccine and filtrate were prepared and given intradermally into the forearms, and the filtrate solution was put into the nares on swabs.

Improvement was immediate and remarkable. At the outset of treatment, she was a sick woman and seemed to be going to pieces. Within a month, with treatments once a week, she seemed to be well and almost free of symptoms. The nasal discharge had almost ceased, as had also cough and expectoration. She was enormously improved. Instead of using many handkerchiefs for the nasal discharge, she only used one handkerchief a day. She was classified as immensely improved, not cured. In 1943, she reported that there was no discharge from the nose but that there was still some cough with sputum. The end-result was good.

L. W., male clerk, aged 23, consulted us in December, 1941, for hemoptysis and chronic sinus disease. We were unable to find evidence of pulmonary tuberculosis, either by x-ray or by physical examination, and the sputum was free of tubercle bacilli on repeated examinations.

He had probably had his sinus condition for seven or eight years, and he had a profuse nasal discharge. Before he consulted us, an x-ray of the head in November, 1941, showed a bilateral chronic maxillary sinusitis. Cultures from both posterior nares yielded Staph. aureus in pure culture. Vacine and filtrate solutions were prepared and given intradermally, and the filtrate solution was put into the nose on cotton swabs. Within a month, with treatments once a week, the patient seemed to be cured. He had no discharge from the nose, and the cough and sputum had ceased. At last report, in 1952, he remained cured.

G. M., an electrician, consulted us in January, 1942, for chronic nasal catarrh, which he had had for some years. X-ray examination of his sinuses showed disease in the left antrum and thickening of the turbinate bones.

Cultures obtained from the posterior nares gave a hemolytic Staph. aureus. Vaccine and filtrate solutions were prepared and given intradermally, and the filtrate solution was put into the nose on swabs. Within a month, with treatments once a week, he was cured, and the discharge from the nose ceased. In 1952, he had remained cured.

M. McC., a 45-year-old schoolteacher, consulted us in June, 1942, for her sinus disease. This had lasted for 15 years. She had always been subject to head colds and polyps during these 15 years. She had had a double antrum operation 11 years before consulting us, with no success.

Since Feb. 1, 1942, she had had head and chest colds with much expectoration. At the time of consultation there was constant discharge from the nose, mostly during the day. Breathing was much more difficult at night. She had always been subject to colds, and had hay fever from the last of August.

An x-ray of the chest showed slight haziness at the apex of the right lung; an x-ray of the sinuses showed clouding of both antra, particularly the right, and also thickening of the turbinate bones.

Materials from both posterior nares grew out a pure culture of hemolytic Staph. aureus. Autogenous vaccine and filtrate solutions were given intradermally, and filtrate solution was put into the nose on swabs. Later, 2 cc. of the filtrate solution was put directly into the antra. When the swabs were used, improvement was slow; when the filtrate was put directly into the antra, improvement was rapid and cure resulted. At the end of treatment, the sense of smell had returned to a large degree. Ten years later, she reported that, though she was much improved, the polyps still bothered her and there was occasional return of the sinus disease.

E. C. Mc., a student, aged 17, was seen by us July, 1943. His sinus disease had begun five years previously, and had continued. The condition resembled a continuous cold, and he would catch cold easily. He had a postnasal discharge in the morning. In 1938, the year the sinus disease began, the left eye bulged so much that the left antrum had to be punctured. In July, 1943, he complained of a constant drainage from the nose; the discharge was sometimes watery and sometimes greenish.

X-ray examination of the lungs showed them to be normal. X-ray examination of the cranial sinuses showed clouding of the left antrum. Cultures from the nose gave a hemolytic Staph aureus. Autogenous vaccine and filtrate solution were given intradermally, and the filtrate solution was put into the nose on swabs. After three doses, at intervals of two weeks, the nasal discharge had almost ceased; at the end of six treatments, there was no discharge from the nose, and the patient seemed to be cured. Later, however, relapse occurred, and he continued to suffer from his sinus disease. Perhaps the filtrate should have been put directly into the antrum.

I. C. C., a 48-year-old mining engineer, was seen by us in September, 1941, complaining of annoying postnasal discharge, which had been present for an indefinite time.

X-ray examination of the cranial sinuses showed clouding of the right antrum, irregular cloudiness of the frontal sinuses, and thickening of the turbinate bones.

Swabs from the posterior nares on culture yielded a hemolytic Staph. aureus and a non-hemolytic Streptococcus. Autogenous polyvalent vaccine and bacterial filtrate were prepared and given intradermally, and the bacterial filtrate was put into the nares on absorbent cotton swabs. There was decided improvement. When, however, the bacterial filtrate was put into the antra, improvement was very marked. The patient coughed much less and had fewer headaches, and the postnasal discharge diminished by 50%. He now has only very slight postnasal discharge in the morning and blows his nose only two or three times. He had no head colds in the winter when he was taking the injections. He was immensely improved, and has had no relapse.

W. McC., a Presbyterian minister, aged 56 years, was seen by us in June, 1943, complaining of deafness and chronic nasal catarrh. The nasal catarrh had lasted for 38 years, having begun when he was 18 years of age, and he was now almost stone-deaf. The postnasal discharge was almost constant. He had been subject to head colds since he was 15 years of age. The nasal septum had been resected, but no appreciable improvement resulted.

X-ray examination of the sinuses showed a partial clouding of the left antrum. Cultures from the postnasal spaces grew a Staph. aureus. Vaccine and bacterial filtrate were prepared and given intradermally; bacterial filtrate was put into the nose on swabs, and improvement immediately occurred. Bacterial filtrate was then, in addition, put into the left antrum, and a very striking result occurred. The postnasal discharge almost ceased, but a slight discharge from the fore part of the nose still persisted. The discharge from the nose lessened to less than one quarter its previous volume. The head colds almost disappeared. As a final result there was slight nasal discharge about three times in 24 hours. The patient was immensely improved but not cured.

In 1952, he suffered relapse, but drainage from the sinuses was so open that he seldom had any pain in the sinus cavities. Head colds were much less frequent and far less severe than formerly.

A. D. C., a 48-year-old merchant, was seen by us in October, 1939, complaining of nasal catarrh. He had had a sore nose since the age of 6 or 7 years. An exostosis developed in the right nostril and had been removed 25 years before consultation. The septum had been punctured at that time. Eight years later, a second operation, for removal of parts of the septum, was done, and since then he had had many head colds and chronic nasal catarrh. He had had sinus disease for the last 15 years.

X-ray examination of the cranial sinuses revealed slight cloudiness of the right frontal sinus. Cultures obtained by absorbent cotton swabs from the posterior nares gave a hemolytic Staphylococcus and a nonhemolytic Streptococcus. Autogeneous polyvalent vaccine and polyvalent filtrate solution were made and given intradermally, and the filtrate was put into the nares on absorbent cotton swabs. He was not improved by treatment.

R. F. L., a 39-year-old merchant, was seen by us in June, 1945, complaining of chronic sinus disease. The sinus trouble had started from 10 to 15 years before. Six months before he consulted us, the left antrum had been punctured, but there had been no benefit from the procedure and the sinus disease was getting worse.

X-ray examination of the sinuses showed a multiple sinus disease involving all the sinuses.

Cultures from the posterior nares showed a hemolytic Staph. aureus. Autogenous vaccine and filtrate solutions were prepared and given intradermally, and the filtrate was put into the nares on absorbent cotton swabs. Immense improvement resulted—at least 75%. At the end of treatment, there was no postnasal discharge and only occasional slight discharge from the left anterior naris. As an end-result, as of 1952, there was partial relapse. The sinus condition completely disappeared, with the exception of some postnasal discharge, necessitating almost constant clearing of the throat. The only time he was bothered by congestion in the sinus was during a head cold.

M. T. S., a stenographer aged 42, was seen by us in November, 1946. Illness had begun about mid August 15 years previously. The nasal discharge disappeared when the frost came. This course was repeated each year five or six years before her visit to us; then the nasal discharge became chronic. When she consulted us the discharge was mostly clear, but with some discoloration; it was constant and very troublesome. She had to sleep on three pillows. She caught colds very easily—had head but not chest colds.

X-ray examination of the sinuses showed all to be relatively clear, with the exception of both frontal sinuses, which showed some clouding consistent with a sinusitis.

Swabs from both posterior nares yielded a pure culture of hemolytic Staph. aureus. Autogenous vaccine and filtrate solutions were prepared and given intradermally, and the filtrate solution was put into the nares on absorbent cotton swabs. At the end of the four weeks, with treatments once a week, she became completely free of symptoms. There was no discharge from the nose, and she could sleep on one pillow; she then used one small handkerchief a day. She contracted a head cold, but it passed off quickly and did not continue indefinitely, as it would have formerly. She was classified as cured.

The end-result as of March, 1952, was still cured. She had remained well and free from symptoms since the end of treatments and had been less subject to colds.

P. E. C., a 34-year-old manufacturer, was seen by us in December, 1946, complaining of postnasal discharge for many years and of repeated sore throats. His tonsils had been removed 20 years previously, and tonsillar tissue again within the past year. He still had sore throats. About 10 years previously he had had an operation on the left side of the nose. There was improvement for a while, but it did not last long. He had had pain over the front sinuses for 10 years. When seen, he had tenderness over the frontal and maxillary sinuses together with a constant discharge from the back of the nose, both day and night.

X-ray examination of the sinuses showed definite clouding of both frontal, the left ethmoid, and the sphenoid sinuses.

Swabs from the posterior nares showed a Staph. aureus and a Staph. albus. Autogenous polyvalent vaccine and filtrate were prepared and given intradermally and the filtrate was put into the nose on absorbent cotton swabs. The odor from the discharge ceased, but the patient did not otherwise improve. He was classified as not improved.

Two years later the sinus disease seemed cured, since there was no postnasal discharge. Six years later, as of March, 1952, the patient had had only one sore throat, in 1951. When last seen, he had very little postnasal discharge and no tenderness over the sinuses.

M. McA., a 38-year-old schoolteacher, consulted us in August, 1947, for her sinus disease, which had begun in 1939. It had followed an attack of scarlet fever in 1938. In 1939, she had a type I pneumonia, and one rib was removed for empyema. The sinus condition remained pretty much the same until the spring of 1947, when her ears started to bother her. The discharge was from both the front and the back of the nose. In the spring of 1947, the discharge became offensive and greatly increased. Since 1939 she had had a morning cough, and asthma accompanied the raising of expectoration. The sputum was heavy and greenish.

X-ray examinations of the sinuses showed the following: The right frontal sinus was undeveloped. The left was well aerated and showed a slight thickening of the mucoperiosteal lining. The maxillary antra showed a slight bilateral clouding, together with a slight, but appreciable thickening of the mucoperiosteal lining. In the ethmoids, there was a bilateral loss of cell wall detail, with evidence of thickening of the mucous membrane lining. The sphenoids showed thickening of the mucous membrane lining.

These changes indicated multiple sinus disease with good drainage, which had progressed to thickening of the mucous membrane lining and a partial destruction of a number of the ethmoid air cells. Both nares contained a hemolytic Staph. albus, and vaccine and filtrate were prepared and given once a week as usual.

At the end of three months there was improvement, but later relapse occurred.

G. W. A., a 69-year-old watchmaker, consulted us in August, 1947, and complained of cough, expectoration, and postnasal drip. The three symptoms had begun one year previously, and had followed influenza. One year before onset of symptoms, he had had polyps and cartilage removed from his nose. During the summer of 1947 asthma had been troublesome.

Findings in the sinuses was as follows: The frontal sinuses showed slight thickening of the mucous membrane lining. In the maxillary sinuses there was slight clouding, bilaterally, but a little more pronounced on the right; also on the right there was seen inferiorly an area of increased density, probably indication of a polyp or a mucocele. The ethmoid sinuses showed the bilateral clouding, more pronounced on the right, associated with thickening of the mucous membrane lining and showed destruction of most of the individual air cells. The sphenoid sinuses appeared normal.

The postnasal discharge was more or less constant and disagreeable. Cultures from the posterior nares gave a nonhemolytic Staph. aureus. Autogenous vaccine and filtrate were prepared. Vaccine was given intradermally, and filtrate put into the nares on absorbent cotton swabs. After eight treatments, given once a week, he was very much better, about 90%, and his sense of smell and taste, which had almost disappeared, returned. He was unable to continue treatment, as he went to Florida for the winter. On arrival in Florida he caught a head cold and did not regain his improved condition. A year later he returned; cultures were made from the nares, and they showed Staph. aureus. Vaccine and filtrate were made and given as before, but no striking gain was seen. With this second treatment he was seen nine times, once a week.

At the end of his first treatments, the drainage from the sinus condition, which necessitated frequent spitting of mucus, had just about ceased. He was able to rest for seven hours without being obliged to get rid of any mucus, whereas formerly he had often been obliged to clear out his throat as often as six or eight times during the night. During the day he had mucus only two or three times.

Chronic Nasal Catarrh

The following patients had no positive evidence by x-ray examination or by transillumination of sinus disease.

Mrs. H., a 32-year-old housewife, consulted us in November, 1937. At the age of 14 she had broken her nose; this was followed by a chronic nasal catarrh. At 15 years of age arthritis had begun, and it had since continued. The arthritis followed the nasal catarrh.

When seen by us, she had a troublesome and constant postnasal discharge. X-ray examination failed to show clouding of any of the sinuses. Cultures from the middle nares yielded (*a*) a nonhemolytic Streptococcus, and (*b*) a Staph. aureus. Autogenous polyvalent vaccine and filtrate solutions were given intradermally, and the filtrate was put into the nares on absorbent swabs. Her arthritis involved almost all her joints, and she was practically confined to bed as a cripple. The arthritic condition was becoming worse. At the end of four months, with treatments once a week, she seemed to be cured. The postnasal discharge had ceased, and the arthritis had disappeared. There was only slight stiffness in the right knee; otherwise the joints were normal. Slight relapse of the arthritis occurred in two years, but with further treatment with vaccine and filtrate the condition disappeared. She remained well, and the nasal condition was normal until her death some years later from pulmonary tuberculosis.

J. H., a 62-year-old accountant was seen by us in April, 1939. He complained of severe postnasal catarrh which became worse when he would lie on his back. The discharge was watery, transparent, and thin, and resembled the white of an egg, but was odorless. He had no pain in the sinuses and no headaches. He had had a perforated septum since childhood. In 1929 he

TABLE 2.—*Results of Treatment in Chronic Nasal Catarrh*

Case	Organisms	Immediate Results	End-Results, 1952
Mrs. H.	Staph. aureus and nonhemolytic Streptococcus	Cured	Cured, no relapse
J. H.	Staph. aureus and nonhemolytic Streptococcus	Cured	13 yr., cured, no relapse
M. P.	Hemolytic Staph. aureus	Cured	6 yr., cured, no relapse
W. A. F.	Hemolytic Staph. aureus, viridans Streptococcus, and hemolytic Streptococcus	Improved	Died, no report
Mrs. McC.	Hemolytic Staph. aureus and hemolytic Streptococcus	Cured	10 yr., cured, no relapse
Mrs. L. E. G.	Hemolytic Staph. aureus	Improved	7 yr., improved, no relapse
C. M.	Staph. aureus	Not improved	.....

had had asthmatic bronchitis, which lasted three years. When we saw him, his catarrh was so severe that he had almost had to give up his business, as he was hardly able to talk because of the discharge dropping into his throat. He used about 12 handkerchiefs in 24 hours.

X-ray examination of the cranial sinuses showed no definite clouding. Swabs from the posterior nares gave cultures of a Staph. aureus and a nonhemolytic Streptococcus. Autogenous vaccine and filtrate were made and given intradermally, and the filtrate was put into nares on absorbent cotton swabs. In four weeks, with treatments once a week, the discharge from the nose had ceased, and instead of using 12 handkerchiefs a day, he used 1 handkerchief every two days. His cure was permanent.

M. P., a 40-year-old woman, was seen by us in November, 1946. She complained of chronic nasal catarrh, which had begun when she was 17 years of age and had since continued. She was very subject to severe head colds with pain over the sinuses. There was a constant dropping from the back of the nose into the throat, and this caused frequent clearing of the throat. The nasal discharge occurred night and day. X-ray examination of sinuses failed to show any abnormalities.

Swabs from both nares gave a hemolytic Staph. aureus. Autogenous vaccine and filtrate were prepared and given intradermally, and the filtrate was put into the nares on cotton absorbent packs. After four treatments, given once a week, she was completely cured and free of all discharge from the nose. There had been no relapse in six years.

W. A. F., a 31-year-old laboratory technician, was seen by us in December, 1939. He was suffering from chronic nasal catarrh. Cultures from the posterior nares showed a viridans

Streptococcus, a hemolytic Staph. aureus, and a hemolytic Streptococcus. Polyvalent autogenous vaccine and filtrates were prepared from these three organisms. The vaccine was given intradermally, and the filtrate was put into the nares on absorbent cotton swabs.

An x-ray examination of the cranial sinuses did not show clouding of any of the sinuses. Treatment was satisfactory, and, although not cured, the patient became 75% improved.

Mrs. McC., a 42-year-old trained nurse, was first seen by us in March, 1942. She complained of cough and expectoration, which had started four years before and which followed a severe chest cold. She had been very subject to chest colds. She also had postnasal catarrh.

X-ray examination of the chest showed no definite abnormal changes, and x-ray of cranial sinuses showed no clouding of any of the sinuses. From the sputum there were grown pure cultures of viridans Streptococcus, hemolytic Streptococcus, Staphylococcus, and nonhemolytic Streptococcus. Autogenous polyvalent vaccine and filtrate were prepared and given as usual once a week, the vaccine intradermally and the filtrate in the nares on absorbent cotton swabs. This was continued for eight months. Great improvement resulted. Cough and sputum were much less, but there was still the postnasal discharge.

Cultures were obtained from the nares and showed hemolytic Staph. aureus and hemolytic Streptococcus. Autogenous polyvalent vaccine and filtrate were prepared and given once a week. The vaccine was given intradermally in the arms, and the filtrate was put into the nares on absorbent cotton swabs. At the end of four months, the discharge from the nose had ceased, and the cough and sputum were slight. The nasal catarrh was cured. Ten years later, in 1952, she reported no nasal drip. She thus had remained cured, had no cough, and had remained free of colds that winter.

Mrs. L. E. G., a 37-year-old trained nurse, was seen by us in February, 1945. She complained of postnasal catarrh, which had lasted for 13 years, since 1932, at which time she had had tonsillitis and a head cold, followed by the postnasal catarrh. She had occasionally had pain in the sinuses. Since 1943, the postnasal discharge had been becoming worse, and she had been having difficulty in breathing through the nose. A pure culture of hemolytic Staph. aureus was grown from both nares, and an autogenous vaccine and filtrate were made. The vaccine was injected intradermally into the forearms, and the filtrate was put into both nares on absorbent cotton swabs.

Treatment lasted for seven months, with visits every week for the first three months and then every two weeks during the next four months. The result of treatment was excellent. At onset the postnasal discharge had been constant, but at the end of treatment the discharge was about six times in 24 hours, and it had decreased greatly in amount. The condition was constantly improving. The end-result was also good. In March, 1952, she reported that there was still some postnasal drainage but that she now rarely had a headache from her sinuses.

C. M., a housewife, was seen by us in December, 1946. She complained of postnasal catarrh, which had begun at 10 years of age and which had since continued. When she was 12 years of age, her tonsils and adenoids were removed. The nasal catarrh had remained about the same, worse in the morning, but present throughout the day, and she had to use many handkerchiefs. The nasal discharge caused frequent cough.

X-ray examination of the sinuses showed them all to be clear, with no evidence of sinusitis. The frontal sinuses were poorly developed and were represented by enlarged supraorbital cells.

Cultures obtained from both nares showed Staph. aureus, and vaccine and filtrate were prepared and given once a week as usual for seven months. The discharge from the nose did not diminish, but became thinner and lost its disagreeable odor. The result was classified as unimproved.

In November, 1947, cultures were obtained from the nares and showed hemolytic Staph. aureus. Vaccine and filtrate were prepared and given as usual for five months, with no definite result.

In March, 1949, cultures were obtained from the nares and showed Staph. aureus and alpha hemolytic streptococcus. Cultures were also taken from the pharynx, and showed Staph. aureus, alpha hemolytic Streptococcus, and Gram-negative bacillus. Polyvalent autogenous vaccine and filtrate were prepared from these three organisms and given as usual. Eight treatments were given, and an excellent improvement resulted, with marked lessening of the nasal discharge.

RESULTS

Immediate Results in Chronic Sinus Disease.—There were 16 patients treated for chronic sinus disease, and 6 of these (37.5%) were cured, that is, at the end of treatment they had no symptoms. Eight more patients were improved, and of these, seven were immensely improved. Thus, 14 of 16 patients were either cured or improved (87.5%). Two patients were not improved (12.5%).

End-Results in Chronic Sinus Disease.—The end or permant results after from 3 to 18 years were as follows. Of the six cured patients, four remained permanently cured (66.7%) and two relapsed (33.3%). Of the eight improved patients, four remained permanently improved, and four relapsed. Thus, of the 14 patients who were either cured or improved, 8 had no relapse (57.14%).

Immediate Results in Chronic Nasal Catarrh.—These patients did not show sinus disease by x-ray examination or by transillumination. Of the seven patients treated, four were cured (57.14%). Two more patients were improved. Thus, six patients were either cured or improved (85.71%). One patient was not improved.

End-Results in Chronic Nasal Catarrh.—The end or permanent results after from 6 to 13 years were as follows. Of the four cured patients, all four remained permanently cured. Of the two improved patients, one remained permanently improved, and one died with no report. Thus, of six patients who were either cured or improved, 5 remained permanently cured (83.3%), and one died with no report.

SUMMARY

The following organisms were found in chronic sinus disease and chronic nasal catarrh.

The Staphylococcus was the organism that was always present in both the sinus disease and the chronic nasal catarrh cases.

The Staph. aureus was always present, both in the sinus disease and the chronic nasal catarrh cases.

The Staph. aureus was present in 13 of the 16 cases of sinus disease.

The Staph. albus was present in three of the sinus disease cases.

The hemolytic Staph. aureus was present in 8 of the 16 sinus disease cases.

A streptococcus was found and given with the vaccines in 4 of 16 sinus disease cases, and was a hemolytic Streptococcus.

The Staph. aureus was present in all six cases of chronic nasal catarrh, and it was a hemolytic Staphylococcus in four of the six cases.

A Streptococcus was present in four of six cases, and in three of the six cases it was a hemolytic Streptococcus.

14 Church St.

DIAGNOSTIC CUTANEOUS REACTIONS, SPECIFIC PREVENTION AND TREATMENT IN EPIDEMIC RESPIRATORY INFECTIONS

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THE IMPORTANCE of cultivatable micro-organisms currently considered as secondary invaders and of the different strains of virus currently considered as primary in the causation of influenza and other epidemic respiratory infections is still a largely unsolved problem. The investigations on influenza virus have yielded information of the greatest importance as regards its presence and nature. The results of virus neutralization tests with convalescent serum for the identification of different strains in epidemics are reminiscent of the early studies on identification of different types of pneumococci by serologic means. At first there was but one type of the Pneumococcus and one strain of influenza virus and then two, three, and so on. The uncertainty as to presence of virus and the type at hand in current epidemic outbreaks, the changes that occur in influenza virus during epidemics, the difficulty and delay entailed in the identification of virus strains, the toxicity and sensitizing properties of virus vaccine made from chick embryo cultures, and the short duration of immunity that follows prophylactic immunization with virus vaccine have proved serious handicaps for the solution of this important problem. Studies from the purely virus standpoint have yielded little of practical value for treatment. The results from the use of antibiotics in the treatment of influenza, as of other epidemic disease due to or associated with virus infections and nonhemolytic streptococci, have also proved disappointing. The need for further information is therefore clearly indicated.

It is the purpose of this communication to review in some detail the existing evidence which bears on this problem and to report the results obtained by the use of new methods—especially those for the detection of specific circulating streptococcal antigen and antibody by the immediate reactions which follow intradermal injection of specific streptococcal antibody and antigen—on the etiology, diagnosis, and specific treatment of epidemic respiratory infections, including influenza.

REVIEW OF PUBLISHED STUDIES

Of the so-called secondary invaders, pneumococci,[1] staphylococci,[2] Bacillus influenzae,[3] and green-producing or nonhemolytic and hemolytic streptococci are by far

From the Department of Bacteriological Research, Longview Hospital.

1. Hirsch, E. F., and McKinney, M.: An Epidemic of Pneumococcus Bronchopneumonia, J. Infect. Dis. **24**:594-617, 1919.

2. Chickering, H. T., and Park, J. H., Jr.: Staphylococcus Aureus Pneumonia, J. A. M. A. **72**:617-626, 1919.

3. Huntoon, F. M., and Hannum, S.: The Rôle of Bacillus Influenzae in Clinical Influenza, J. Immunol. **4**:167-187, 1919.

the most important. Streptococci (including a pandemic strain [4] during the pandemic of influenza of 1918), especially of the alpha type, have been isolated consistently by various investigators [5] and by me from persons stricken,[6] from indoor and outdoor air,[7] from milk supplies,[8] from casual water and water supplies [9] during epidemics which have occurred chiefly in winter months since 1918 and by myself [10] and others [11] in studies of primary atypical or so-called "virus" pneumonia. The nonhemolytic streptococci isolated by the special methods used from persons ill during epidemics of respiratory infections and from air, milk, and water were shown to possess pneumotropic virulence which was roughly proportional to the severity of epidemics. The nonhemolytic streptococci isolated during the pandemic of 1918 were extremely virulent. Intratracheal insufflation in guinea pigs caused death in high incidence from widely disseminated areas of extreme emphysema, atelectasis, hemorrhagic edema, tracheitis, bronchitis, bronchopneumonia, and necrosis of alveolar epithelium and capillaries. These findings simulated the extraordinarily severe clinical, pathological, and histological picture of the naturally occurring disease in human beings during the pandemic.[12]

The nonhemolytic streptococci were isolated consistently, since in studies of milder epidemics of respiratory infections, including the common cold and "virus" pneumonia, they had a similar predilection for the respiratory tract on cerebral, nasal, and intraperitoneal inoculation in mice, guinea pigs, and rabbits, respectively. The symptoms and lesions were less pronounced and the mortality far lower than during

4. Rosenow, E. C.: Studies in Influenza and Pneumonia: III. The Occurrence of a Pandemic Strain of Streptococcus During the Pandemic of Influenza, J. A. M. A. **72**:1608-1609, 1919.

5. Blanton, W. B., and Irons, E. E.: A Recent Epidemic of Acute Respiratory Infection at Camp Custer, Mich.: Preliminary Laboratory Report, J. A. M. A. **71**:1988-1991, 1918. Falk, I. S.; Harrison, R. W.; McKinney, R. A., and Stuppy, G. W.: Experiments on the Etiology of Influenza: Preliminary Report, ibid. **93**:2030-2031, 1929. Weisner, R. R.: Streptococcus Pleomorphus und die sogenannte spanische Grippe, Wien. Klin. Wchnschr. **31**:1101-1104, 1918. Rosenow, E. C.: Studies in Influenza and Pneumonia: II. The Experimental Production of Symptoms and Lesions Simulating Those of Influenza with Streptococci Isolated During the Present Pandemic, J. A. M. A. **72**:1604-1608, 1919. Mathers, G.: The Bacteriology of Acute Epidemic Respiratory Infections Commonly Called Influenza, J. Infect. Dis. **21**:1-8, 1917.

6. Rosenow, E. C.: Studies on the Etiologic Relation of Streptococci to Acute Epidemic Respiratory Infections, Am. J. Clin. Path. **15**:319-333, 1945.

7. Rosenow, E. C.: Isolation from the Air of Streptococci and Streptococcal Antigens Resembling Those Associated with Certain Epidemic Diseases, J. Bact. **39**:73-74, 1940.

8. Rosenow, E. C.: Isolation from Milk Supplies of Specific Types of Green-Producing (Alpha) Streptococci and Their Thermal Death Point in Milk, Minnesota Med. **27**:550-556, 1944.

9. Rosenow, E. C.: Specific Types of Alpha Streptococci and Streptococcal Antigen in Unpotable Water and Water Supplies, Am. J. Clin. Path. **15**:513-528, 1945.

10. Rosenow, E. C.: Diagnostic Cutaneous Reactions to Intradermal Injection of Natural and Artificial Antibody and of Antigen Prepared from Streptococci Isolated in Studies of Diverse Diseases, Ann. Allergy **6**:485-496, 1948.

11. Thomas, L.; Mirick, G. S.; Curren, E. C.; Ziegler, J. E., Jr., and Horsfall, F. L., Jr.: Serological Reactions with an Indifferent Streptococcus in Primary Atypical Pneumonia, Science **98**:566-568, 1943.

12. LeCount, E. R.: The Pathological Anatomy of Influenzal Bronchopneumonia, J. A. M. A. **72**:650-652, 1919; Disseminated Necrosis of Pulmonary Capillaries in Influenzal Pneumonia, ibid. **72**:1519-1520, 1919. Bell, E. T.: The Pathology of the Lungs in Pneumonia Following Influenza, Journal Lancet **39**:3, 1919.

the pandemic, paralleling the severity of the disease of the epidemics in question. Moreover, the nonhemolytic streptococci on isolation in dextrose-brain broth from sputum and nasopharyngeal swabbings in studies of the different epidemics were found to have characteristic distribution curves of cataphoretic velocity or electrical potential.[13] Intradermal injection of solutions of the euglobulin fraction of the serum of horses that had been immunized with the nonhemolytic Streptococcus from influenza caused an immediate erythematous reaction indicating specific streptococcal antigen in skin or blood and hence a corresponding streptococcal infection in influenza and in influenzal and "virus" pneumonia.[6] The agglutinative titer of the serum of persons with influenza and influenzal and "virus" bronchopneumonia for the respective streptococci increased as recovery occurred.[14] Antistreptococcus serums were prepared in horses with the streptococci in which the antigenic specificity was preserved at 10 C. throughout the long period of immunization by partial dehydration of the freshly isolated strains in dense suspension (1,000,000,000,000 organisms per cubic centimeter) of glycerin (2 parts) and saturated NaCl solution (1 part). Such antiserums agglutinated the streptococci and precipitated specifically the antigen in NaCl solution washings of nasopharyngeal swabbings and in cleared suspensions of the streptococci whose specificity had been preserved in the glycerin-NaCl solution menstruum in interface precipitation tests.[15]

The high degree of protection afforded by prophylactic immunization in advance of anticipated epidemics or abreast with epidemics with vaccines containing a high percentage of the nonhemolytic Streptococcus whose specificity had been preserved in the glycerin NaCl solution menstruum further indicates the importance of bacterial infection in influenza. The protection afforded by three weekly subcutaneous injections of the vaccine and then one injection at three- or four-week intervals during epidemics was most striking. The incidence of influenzal attacks, the dreaded hemorrhagic edema and bronchopneumonia, miscarriages in pregnancy, and deaths from hemorrhagic edema and bronchopneumonia during the pandemic were from three- to eightfold lower in the vaccinated groups than in the control unvaccinated groups.[16] Persons having chronic bronchitis, sinusitis, myositis, or infectious arthritis

13. Rosenow, E. C.: (*a*) Cataphoretic Velocity and Virulence of Streptococci Isolated from Throats of Human Beings, from Raw Milk, Flies, Water, Sewage and Air During Epidemics of the Common Autumnal Cold, Am. J. Hyg. **19**:1-21, 1934; (*b*) Cataphoretic Time and Velocity of Streptococci and Pneumococci: Studies on Organisms Isolated in Cases of the Common Cold, Influenza, Bronchopneumonia and Lobar Pneumonia, J. Infect. Dis. **54**:91-122, 1934; (*c*) Cataphoresis as a Control of the Specificity of Streptococcal Vaccines: Influenzal Streptococcus Vaccine in the Prevention and Treatment of Infections of the Respiratory Tract, J. Immunol. **26**:401-433, 1934; (*d*) footnote 6.

14. Rosenow, E. C.: Studies in Influenza and Pneumonia: X. The Immunologic Properties of the Green-Producing Streptococci from Influenza, J. Infect. Dis. **26**:597-613, 1920.

15. Rosenow, E. C.: Demonstration of the Association of Specifically Different Alpha Streptococci with Various Diseases, and Methods for the Preparation and Use of Specific Antiserums and Vaccines in Diagnosis and Treatment, Am. J. Clin. Path. **12**:339-356, 1942.

16. (*a*) Rosenow, E. C., and Sturdivant, B. F.: Studies in Influenza and Pneumonia: IV. Further Results of Prophylactic Inoculations, J. A. M. A. **73**:396-401, 1919. (*b*) Rosenow, E. C.: Observations on the Cause and Prevention of Influenza and Influenzal Pneumonia, Minnesota Med. **12**:366-368, 1929. (*c*) Rosenow, E. C., and Osterberg, A. E.: A Method for the Preparation of Prophylactic and Autogenous Lipovaccines, J. A. M. A. **73**:87-91, 1919. (*d*) Rosenow, E. C., and Heilman, F. R.: Streptococcal Vaccines in the Prevention and Treatment of Respiratory Infections: A Clinical and Experimental Study, Am. J. Clin. Path. **8**:17-27, 1938.

to whom the vaccine was given also benefitted.[17] Others reported similar results.[18] Having found a method for preserving antigenic specificity and for reducing toxicity of the highly mutable nonhemolytic Streptococcus, an attempt was made to find a vaccine sufficiently nontoxic and broad in antigenicity that would afford protection not only for the common cold but also for the severer influenzal infections. Two vaccines were used, at first one prepared from the streptococci isolated in studies of the common cold and one containing the streptococci from patients with influenza. It was found that the former protected against the common cold but not against influenza. The latter afforded protection geographically against both the common cold and influenza.[19] Throughout these studies the vaccine was used also in treatment. Results obtained by us and by the large numbers of physicians to whom it was sent gratis for study were also uniformly favorable geographically.

Protective and curative action of the vaccine in animals was similar to that obtained in human beings,[20] and the monovalent antistreptococcal serum had curative action in persons having influenza due to serologically homologous nonhemolytic streptococci.

It should be emphasized that the results obtained in the studies reviewed are attributable to the special methods employed. The characteristic virulent and nonvirulent nonhemolytic streptococci, morphologically indistinguishable, were found in mixture in nasopharyngeal swabbings and in sputum. The former had a distinctive influenzal distribution curve; the latter had a normal distribution curve of cataphoretic velocity or electrical potential. The former was highly sensitive to oxygen and usually did not grow on blood agar in primary aerobic cultures; whereas the latter grew in large numbers in mediums affording a gradient or reduced oxygen tension and other conditions favorable for growth, such as 0.2% dextrose broth to which pieces of fresh calf brain (1 part to 6 or 7 parts of the broth) in tall columns were added before autoclaving. The virulent nonhemolytic streptococci in dextrose-brain broth outgrew the nonvirulent variants in young cultures. The use of this medium for primary isolations and for rapidly repeated subcultures for inoculation of animals and the preservation of antigenic specificity in the glycerin and sodium chloride solution menstruum for serologic studies and for the preparation of vaccines was found essential. The nonhemolytic Streptococcus having high pneumotropic virulence, as that isolated during the pandemic, commonly changed to hemolytic type in vivo and in vitro, and the hemolytic variant often reverted to nonhemolytic type. A rise and fall in severity of symptoms and mortality occurred in family groups and during successive epidemic waves during the pandemic and was shown to be due to changes in virulence of the Streptococcus. Similar changes in severity of the infection and mortality occurred on successive intratracheal guinea pig passage of the causative nonhemolytic Streptococcus.[21]

17. Rosenow,[13b] Rosenow and Sturdivant.[16a]

18. Fennel, E. A.: Prophylactic Inoculation Against Pneumonia: A Brief History and Present Status of Procedure, J. A. M. A. **71**:2115-2120, 1918. Cecil, R. L., and Vaughan, H.: Results of Prophylactic Vaccination Against Pneumonia at Camp Wheeler, J. Exper. Med. **29**:457-483, 1919. Moody, E., and Howard, W. M.: Control of Juvenile Asthma by Vaccine Therapy: Report of Small Series, Arch. Pediat. **58**:774-778, 1941.

19. Footnote 13*c*. Footnote 6. Footnote 16*d*.

20. Rosenow,[6] Footnote 13*c*.

21. Rosenow, E. C.: Studies in Influenza and Pneumonia: IX. Changes in the Green-Producing Streptococcus Induced by Successive Animal Passage and Their Significance in Epidemic Influenza, J. Infect. Dis. **26**:567-596, 1920.

The common occurrence of gastroenteritis during the course of epidemics of influenza and other respiratory infections is well known. Nonhemolytic streptococci having predilection for the gastrointestinal tract in animals have been consistently isolated from the nasopharynx and stools of persons so afflicted and often from the water supply.[22] Changes from pneumotropic to gastroenterotropic type of the Streptococcus have occurred on successive passage through animals,[22] and the occurrence of both the respective streptococci and the viruses in epidemics of influenza has been reported.[23] The occurrence of lethargic and other forms of encephalitis following epidemics of influenza is also well known. Most important, the strains of "neurotropic" nonhemolytic streptococci isolated in studies of encephalitis lost "neurotropic" virulence and acquired "pneumotropic" virulence, causing pneumonitis on successive cerebral passage through animals.[24] The results of these experiments indicate that the older classifications of influenza as "catarrhal," "brain," and "gastrointestinal" fever, respectively, were appropriately designated by these descriptive terms. Nonhemolytic streptococci having distinctive elective localizing properties were isolated respectively from these three disease entities, and in each of these—influenza and other epidemic respiratory infections, encephalitis, and gastroenteritis—characteristic viruses having respective specific disease-producing properties have been identified.

On the basis of facts such as these it was postulated that the respective streptococci and viruses may be related and that the respective specificities may be due to mutation or dissociation. A long series of experiments were made to determine the validity of such a hypothesis. The three respective filtrable agents resembling the natural viruses have been produced experimentally from the respective "neurotropic," [25] "enterotropic," [22a] and "pneumotropic" [26] green-producing or nonhemolytic streptococci. The strains of streptococci in each instance were so far removed from the original source as to eliminate the presence of the respective natural viruses.

The results of studies on the relation of pneumotropic streptococci to influenza virus were especially relevant. Pneumotropic nonhemolytic streptococci were isolated by the special methods employed from natural influenza virus and from the experimentally produced filtrable pneumotropic agents resembling virus. The nonhemolytic streptococci isolated from the nasopharynx of persons having influenza, from the experimentally produced virus, and from natural virus all produced pneumonitis on nasal inoculation in mice. Mice were immunized against the natural and the

22. Rosenow, E. C.: (*a*) Infectious Gastro-Enteritis: An Epidemiologic and Laboratory Study, Am. J. Digest. Dis. **11**:381-391, 1944; (*b*) Studies in Influenza and Pneumonia: VIII, Experiments on the Etiology of "Gastro-Intestinal" Influenza, J. Infect. Dis. **26**:557-566, 1920.

23. Rosenow, E. C.: Streptococci and Diplostreptococci and the Respective "Viruses" in the Etiology and Epidemiology of Epidemic Respiratory Infections and Infectious Gastroenteritis, Am. J. Digest. Dis. **17**:261-270, 1950.

24. Rosenow, E. C.: Changes in the Streptococcus from Encephalitis, Induced Experimentally, and Their Significance in the Pathogenesis of Epidemic Encephalitis and Influenza, J. Infect. Dis. **33**:531-556, 1923.

25. (*a*) Kendall, A. I.: Observations upon the Filterability of Bacteria, Including a Filterable Organism Obtained from Cases of Influenza: Studies in Bacterial Metabolism, Science **74**:129-139, 1931. (*b*) Rosenow, E. C.: Studies on the Virus Nature of an Infectious Agent Obtained from 4 Strains of "Neurotropic" Alpha Streptococci, J. Nerv. & Ment. Dis. **100**:229-262, 1944; (*c*) The Relation of Neurotropic Streptococci to Encephalitis and Encephalitic Virus, Proc. Staff Meet., Mayo Clin. **17**:551-560, 1942.

26. Rosenow, E. C.: Studies on the Relation of Pneumotropic Streptococci to Influenza Virus, Am. J. Clin. Path. **15**:362-380, 1945.

experimental virus with the influenzal streptococcic vaccine. Natural and experimental viruses were neutralized in parallel by influenzal antistreptococcic serum and by convalescent serum.[22a] In support of the results of these studies, it has since been shown with the electron microscope that the virus of influenza consists of small spheres, ovoids singly and doubly and in short and long filaments or in chain formation,[27] suggesting further that the Streptococcus and virus are related and antigenically similar.

Having reviewed the studies which bear so directly on the problem involved, I shall next report the results of further studies by new methods which further indicate the importance of nonhemolytic streptococci in influenza.

TABLE 1.—*Results in Mice After Cerebral and Nasal Inoculation of the Streptococcus Isolated from Nasopharynx of Persons Having Epidemic Respiratory Infection, from Outdoor Air During Epidemic Prevalence of Such Infection, and as Controls from Poliomyelitis and Persons Not Having Respiratory Infections*

Source and Type of Material Inoculated			Strains	Mice				Streptococci from			
				Inoculated	Percentage			Lungs		Brain	
					That Died	In which		Cultured	% Plus	Cultured	% Plus
						Polioencephalitis Developed	Pneumonitis Developed				
Streptococci from	Nasopharynx of persons having epidemic respiratory infection		70	131	31	10	[48]	40	[80]	38	53
	Poliomyelitis	Nasopharynx of persons stricken	18	81	39	[54]	16	9	22	33	[69]
		Virus from monkey and mouse	8	88	27	[47]	9	11	27	37	[70]
	Well persons or persons ill not with respiratory infection		69	156	17	12	14	10	30	18	15
Outdoor air during general prevalence of epidemic respiratory infection		Streptococci*	1	33	24	24	[64]	19	[78]	17	18
		Filtrates*	1	18	0	0	[39]	12	[50]	17	18

RESULTS OF FURTHER STUDIES BY NEW METHODS

The results in control material and in mice, following inoculation of nonhemolytic streptococci isolated in dextrose-brain broth and obtained from the nasopharynx of well persons and of persons having respiratory infection and from the NaCl solution washings of absorbent material exposed to outdoor air on an automobile during a drive of 1,850 miles in November, 1950, through regions where respiratory infections were generally prevalent, are summarized in Table 1. While under ether anesthesia, the mice were inoculated cerebrally with 0.03 ml. and nasally with 0.06 ml. of 10: 1 suspension in sodium chloride solution of pure cultures of the nonhemolytic streptococci from the end-point of growth of serial dilution cultures in dextrose-brain broth. Mice that died were examined as soon after death as possible. Those that survived

27. (*a*) Taylor, A. R.; Sharp, D. G.; Beard, D.; Dingle, D. H., and Feller, A. E.: The Isolation and Characterization of Influenza A Virus (PR8 Strain), J. Immunol. **47**:261-282, 1943. (*b*) Murphy, J. S.; Karzon, A. T., and Bang, F. G.: Studies of Influenza (PR8) Infected Tissue Cultures by Electronmicroscopy, Dept. Med., The Johns Hopkins Univ. School of Med., Baltimore, Md.

were killed with ether three to five days following inoculation. A high degree of specificity of the respective strains is shown by the incidence of lesions and isolations of streptococci from the lesions produced.

In order to determine further the epidemiologic importance of the Streptococcus isolated from the sampling of outdoor air and as a control, NaCl solution and a Seitz filtrate of NaCl solution washing of the exposed material were nebulized into sterilized gallon jars, into each of which five mice were placed. Into each of two of these jars was nebulized 5 ml. of a dextrose-brain broth culture isolated from the filtrate of sodium chloride solution washings of the exposed material. Into each of two other jars was nebulized the Seitz filtrate of such washings and, as a control, 5 ml. of sterile sodium chloride solution was nebulized into each of two jars. The mice were killed with ether on the third day after the nebulization. Four of the 10 that were made to respire the Streptococcus revealed slight to severe pneumonitis, and the Streptococcus was isolated from affected lungs. Five of the 10 that respired the filtrate of NaCl solution washings of material which had been exposed to outdoor air revealed pneumonitis, and the Streptococcus was isolated. The lungs of the 10 control mice were normal, and cultures for streptococci were negative.

Through studies on the in vitro production of antibodies, new materials suitable for diagnosis and specific treatment have become available.[28] When suspensions in sodium chloride solution containing 20,000,000,000 streptococci per milliliter prepared from dilutions of the dense glycerin-sodium chloride solution suspension are autoclaved at 17-lb. (7.7 kg.) of pressure for 96 hours, the organisms disintegrate; the remnants become sharply agglutinated and brownish in color, and substances resembling the natural antibody suitable for diagnostic cutaneous tests and treatment appear in the supernatant sodium chloride solution.

Erythematous reactions to intradermal injection of 0.03 ml. of the supernatant diluted with equal parts of NaCl solution containing 0.4% phenol have been found to serve as a measure of specific antigen in skin or blood and hence of a corresponding streptococcal infection. Erythematous reactions to the bacteria-free supernatant of streptococci containing 10,000,000,000 streptococci per milliliter after heating at 70 C. for one hour and similarly injected intradermally serve as a measure of antibody in skin or blood. Antibody produced by autoclaving suspensions containing 10,000,000,000 streptococci per milliliter for three hours after adding 1.5% hydrogen peroxide has been found to have high specific agglutinative titers and on subcutaneous therapeutic injection, after bringing the pH to 6.5, has been found to cause a sharp drop in antigen and rise in antibody on repeat intradermal tests and often a corresponding improvement in symptoms. Therapeutic response and diminution of cutaneous reaction occur also following injection of the supernatant of suspensions autoclaved for 96 hours without hydrogen peroxide.

The results of cutaneous tests in groups of persons having respiratory infections and in well control persons according to season for four consecutive years and in persons having acute and chronic sinusitis, bronchial asthma, and hay fever are summarized in Table 2. The average reactions indicating "respiratory" streptococcic antigen and antibody to intradermal injection of antibody and antigen respectively were uniformly far greater in persons having respiratory infections than in persons

28. Rosenow, E. C.: Production in Vitro of Substances Resembling Antibodies from Bacteria, J. Infect. Dis. **76**:163-178, 1945; Studies on the Nature of Antibodies Produced in Vitro from Bacteria with Hydrogen Peroxide and Heat, J. Immunol. **55**:219-232, 1947.

TABLE 2.—*Intradermal Antibody-Antigen and Antigen-Antibody Reactions in Persons Having Acute Respiratory Infection and Well Persons According to Season and in Persons Having Sinusitis, Bronchial Asthma, and Hay Fever or Allergic Rhinitis, Respectively*

Year	Season	Acute epidemic respiratory infection + or 0	Cutaneous reactions (sq.cm.) to intradermal injection of thermal antibody and antigen prepared from streptococci isolated in studies of											
			Epidemic respiratory infections			Chronic Encephalitis		Arthritis		Well persons		Control - Pneumococci Types I, II, III	
			Indicating in skin or blood respective streptococcal										
			Persons Tested	Antigen	Antibody	Persons Tested	Antigen	Persons Tested	Antigen	Persons Tested	Antigen	Persons Tested	Antigen
1947	Summer & Autumn	+	77	12.03	7.02			77	2.66				
		0	121	3.77	2.92	104	2.44	45	3.11				
1948	Spring & Autumn	+	60	12.67	7.45	9	4.60	33	2.43	16	1.34		
		0	165	4.02	2.19	94	2.59	54	2.59	45	2.21		
1949	Autumn & Winter	+	96	13.97	9.17	55	4.25	20	3.96	16	2.42	10	5.52
		0	124	8.61	6.29	124	1.10	124	1.87	16	3.42	10	3.51
1950		+	67	13.48	7.95	11	3.64	21	2.41			25	3.22
		0	119	7.09	5.26	40	2.92	18	1.27	23	1.67	58	2.99
Sinusitis		Acute	7	16.81	6.02	6	3.88	6	3.45				
		Chronic	16	7.34	5.43	7	4.40	16	2.10				
Bronchial Asthma			11	11.08	8.40	4	3.68	10	2.63				
Hay Fever and Allergic Rhinitis			22	6.93	1.14	14	2.90						

TABLE 3.—*Intradermal Antibody-Antigen and Antigen-Antibody Reactions Indicating Antigen and Antibody, Respectively, in Skin or Blood in Well Persons and Persons Having Respiratory Infection in Relation to the Seasonal Incidence of Respiratory Infection and Poliomyelitis*

Persons studied according to disease and season			General incidence of		Groups	Persons Tested	Erythematous reactions (sq.cm.) indicating streptococcal antigen and antibody in skin or blood on intradermal injection respectively of thermal antibody and of antigen prepared from streptococci isolated in studies of					
							Res. Infection		Poliomyelitis		Controls	
			Resp. Infection	Poliomyelitis			Antigen	Antibody	Antigen	Antibody	Antigen	Antigen
Persons Tested Twice While	Well	Sept. 1948	Mod.	Mod.	4	99	6.61	4.29	11.32	8.32	2.12	2.34
		Feb. 1949	High	0			11.21	7.54	4.55	4.30	4.18	3.08
	Having Resp. Infection	Sept. 1948	Mod.	Mod.	4	79	12.77	5.52	12.55	8.35	3.99	3.30
		Feb. 1949	High	0			13.53	7.79	5.80	4.14	4.42	2.46
Persons Tested Once While	Well	May 1948	Mod.	0	1	78	6.46	5.20	3.26	2.85	3.41	1.88
		Sept. 1948	0	Mod.	8	383	4.86	3.52	11.77	8.79	1.66	1.24
		Feb. 1949	High	0	5	103	10.46	7.70	5.94	3.62	4.29	4.98
	Having Resp. Infection	May 1948	Mod.	0	1	35	13.91	6.70	4.05	3.50	3.57	1.55
		Sept. 1948	Mod.	Mod.	7	101	12.14	5.80	12.14	8.55	3.47	2.30
		Feb. 1949	High	0	5	60	15.17	9.45	5.20	5.47	4.94	4.24

having encephalitis or arthritis and in well persons. Reactions indicating "respiratory" antigen and antibody both in ill persons and in well persons were uniformly significantly greater during autumn and winter in 1949 and 1950 than during summer and autumn in 1947 and during summer in 1948 (not shown in the Table), corresponding to the usual seasonal incidence and severity of respiratory infections. Reactions to control antibody indicating antigen were uniformly far less, and there were no significant seasonal changes. The reactions indicating infection by the "respiratory" type of Streptococcus in sinusitis, bronchial asthma, and hay fever parallel roughly the clinical and bacteriological findings in these conditions.

The results of similar tests made in a large number of well persons and persons having respiratory infections in relation to the seasonal incidence of respiratory infection and poliomyelitis, respectively, are summarized in Table 3. Cutaneous reactions indicating "respiratory" streptococcic antigen and antibody in well persons but especially in persons having respiratory infection were greatest during winter months, when epidemics of influenza and other respiratory infections were prevalent.

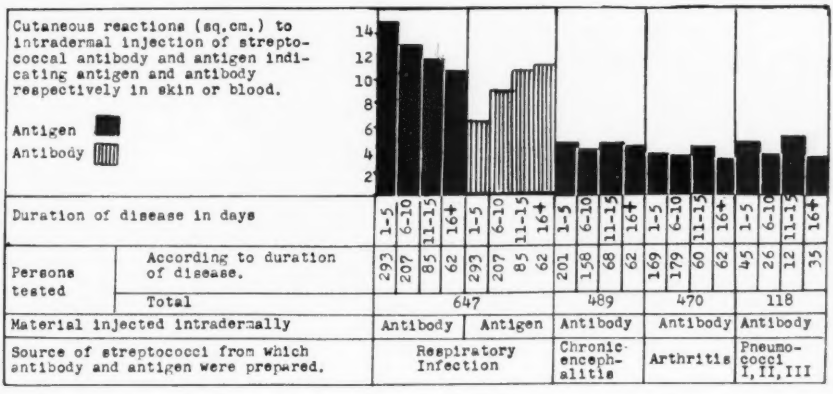
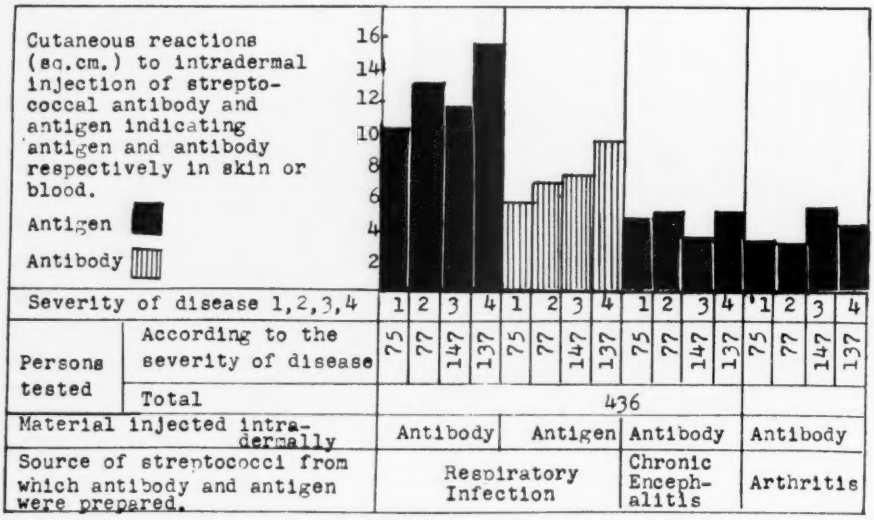


Chart 1.—Cutaneous reactions to intradermal injection of "respiratory" streptococcal antibody and antigen and in contrast to those of control antibody in persons having acute respiratory infection, according to the duration of the disease.

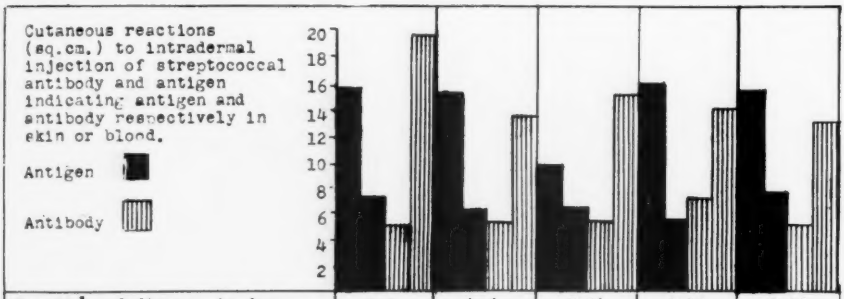
In sharp contrast, "respiratory" antigen and antibody were lowest and reactions indicating "poliomyelitis" streptococcic antigen and antibody were uniformly greatest in summer, when poliomyelitis occurs in highest incidence. Reactions to intradermal injection of the control antigen and antibody were uniformly far less and did not change according to season. The large number of persons tested and summarized in Tables 2 and 3 consisted of widely separated groups, chiefly nurses, college students, physicians, and personnel in hospitals in Ohio, Illinois, Wisconsin, and Minnesota.

The results of cutaneous reactions in a large number of controls and persons having respiratory infections, according to the duration of the disease, are shown graphically in Chart 1. Cutaneous reactions to the respiratory thermal antibody indicating streptococcal antigen in persons having respiratory infections were greatest at the onset of the disease and diminished with time as recovery ensued. Reactions to antigen indicating antibody were least pronounced at the onset and increased as recovery occurred. Reactions to the control antibody prepared respectively from streptococci isolated in studies of encephalitis and arthritis and pneumococci in pneumonia were minimal throughout.



Severity of disease 1,2,3,4		1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
Persons tested	According to the severity of disease	75	77	147	137	75	77	147	137	75	77	147	137	75	77	147	137
	Total	436															
Material injected intradermally		Antibody				Antigen				Antibody				Antibody			
Source of streptococci from which antibody and antigen were prepared.		Respiratory Infection								Chronic Encephalitis				Arthritis			

Chart 2.—Cutaneous reactions to intradermal injection of thermal "respiratory" streptococcal antibody and antigen and in contrast to those of control antibody in persons having acute respiratory infection, according to the severity of the disease.



Duration of disease in days		1-3				4-6				7-14				3-10				7-12			
Material injected intradermally		Antibody		Antigen		Antibody		Antigen		Antibody		Antigen		Antibody		Antigen		Antibody		Antigen	
Tests in relation to therapeutic injection of "respiratory" streptococcal thermal antibody. Before, 24 hours after.		Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After
Persons tested.		21				17				9				6				6			
Diagnosis		Acute Respiratory Infection												"Virus" Pneumonia				Acute Sinusitis			

Chart 3.—Cutaneous reactions in relation to subcutaneous therapeutic injection of thermal antibody prepared from streptococci isolated in studies of respiratory infection in persons having acute respiratory infection, "virus" pneumonia, or acute sinusitis.

The results of cutaneous reactions in persons having respiratory infection, according to severity of attacks, are summarized similarly in Chart 2. It will be noted that reactions indicating streptococcal antigen and antibody were directly proportional to the severity of the disease, but reactions indicating antibody were proportionately far less than those indicating antigen. Reactions to the control antibody were minimal.

The results of cutaneous reactions in response to subcutaneous therapeutic injection of "respiratory" streptococcal thermal antibody in three groups of persons having acute respiratory infection and one group each having "virus" pneumonia and acute sinusitis are shown graphically in Chart 3. It will be seen that a sharp drop in cutaneous reactions to repeat intradermal injection of thermal antibody, indicating circulating antigen, and a great increase in reactions to repeat intradermal injection of antigen, indicating antibody, often associated with a corresponding clinical improvement, occurred 24 hours after therapeutic injection of antibody. The diminution in antigen and increase in antibody were greatest during the early stages of the disease.

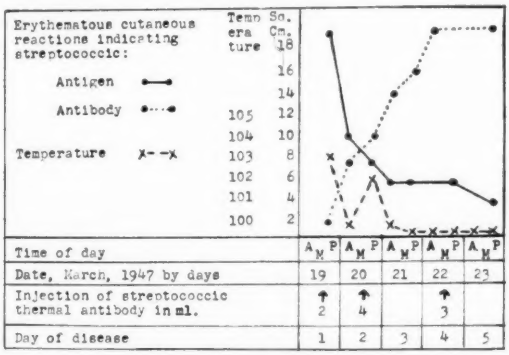


Chart 4.—Effects of therapeutic injection of influenzal streptococcic thermal antibody as indicated by cutaneous reactions to intradermal injection of thermal antibody and antigen, respectively, and temperature in a case of acute influenza.

The effect of therapeutic injection of thermal antibody prepared from streptococci isolated in studies of influenza and other respiratory infections is well illustrated in the following case.

The patient, a business man of robust health, 45 years of age, awoke March 19, 1947, with a sore throat but went to work as usual. The sore throat grew worse, as a dry cough, pain in the chest, and a chill developed during the afternoon. When seen at his home later that afternoon, he had a flushed face, ached severely, and felt exhausted. His temperature was 102.8 F. The blood leucocyte count was 6,500. The nasopharynx, uvula, and soft palate were congested and edematous. The chest was normal. The nasopharynx was swabbed. A pure culture of a greening streptococcus was isolated from the end-point of growth, 10^{-10} of serial dilution cultures in dextrose-brain broth, which on nasal inoculation produced pneumonitis in mice in high incidence. Two milliliters of the respiratory streptococcal antibody was injected subcutaneously. He felt quite well the next morning, went to work, and made a prompt and complete recovery without postinfluenzal exhaustion and was given two additional subcutaneous injections of thermal antibody. The erythematous reactions to intradermal injection of thermal antibody indicating "pneumotropic" streptococcal antigen and intradermal injection of streptococcal antigen indicating antibody in skin or blood, the concomitant drop in temperature, and the time and amounts of subcutaneous injection of thermal antibody are shown graphically in Chart 4.

During an institutional epidemic of influenza it was found that penicillin had little or no curative action in persons in whom the cutaneous test indicating antigen was high, and the reduction of antigen and increase in antibody to repeat tests were far less than in a control group not receiving penicillin, and improvement in symptoms for reasons still obscure was correspondingly delayed. The cutaneous tests have proved of great value in diagnosis of the type of infection at hand, and repeat tests have been of great value in determining the need for reinjection of antibody in treatment.[29]

SUMMARY AND CONCLUSIONS

The results of bacteriological and viral studies of influenza and other epidemic respiratory infections are reviewed, and results of further studies on etiology and treatment by new methods are reported.

The nonhemolytic or green-producing alpha Streptococcus was isolated consistently by special methods from the nasopharynx of persons stricken during life and from the pneumonic lungs after death during the pandemic of 1918-1919 and from the nasopharynx in recurring epidemics ever since. Culturally, morphologically, and antigenically the Streptococcus isolated throughout the years was identical or very similar, but pneumotropic virulence varied according to severity of the disease and epidemic. It was isolated in significant incidence from filtrates of nasopharyngeal washings, from natural influenza virus, from pneumonic lungs produced experimentally in mice with naturally and experimentally produced virus from which the Streptococcus had been isolated, from allantoic fluid of embryonated chicken eggs that had been inoculated with natural influenza virus, and from washings of absorbent material that had been exposed to outdoor air during epidemics of influenza and from filtrates of such washings. Realizing the importance of these experiments, special precautions in the making of these cultures were taken to prevent contamination from extraneous sources.

The importance of pneumotropic nonhemolytic streptococci is indicated furthermore by the formation of agglutinins specific for the Streptococcus during recovery from epidemic influenza, by the formation of specific agglutinin and precipitin and protective antibodies in the serum of horses immunized with the Streptococcus, by the high degree of protection afforded by prophylactic immunization of human beings with the heat-killed streptococcal vaccine containing the nonhemolytic Streptococcus freshly isolated in mediums, such as dextrose-brain broth, which furnish a gradient of oxygen tension and in which specificity was maintained on storage at 10 C. of the partially dehydrated streptococci in dense suspensions of glycerin (2 parts) and saturated NaCl solution (1 part).

Pneumotropic filtrable agents resembling influenza virus have been produced experimentally from the streptococcus isolated respectively from persons with influenza and from natural influenza virus.[30] Convalescent serum and antistreptococcal serums produced in horses, with the Streptococcus neutralized, parallel the natural and experimental influenza virus, and mice were successfully immunized against

29. (*a*) Rosenow, E. C.: Nonhemolytic Streptococci in Relation to an Epidemic of Influenza: Diagnostic Cutaneous Tests and Specific Treatment, A. M. A. Arch. Otolaryng. **54**:609-619, 1951; (*b*) Studies in Influenza and Pneumonia: Therapeutic Effects of a Monovalent Antistreptococcus Serum in Influenza and Influenzal Pneumonia, J. Infect. Dis. **26**:614-622, 1920.

30. Rosenow,[25b] Footnote 26.

inoculations of natural and experimental virus with composite streptococcic vaccines used successfully for prevention and treatment of influenza in human beings.

Relationship of the Streptococcus and the virus is indicated further (*a*) by the production in animals of pneumonitis with both the Streptococcus and the virus, (*b*) by specific agglutination of the pneumotropic Streptococcus and by neutralization of the virus by convalescent serum, (*c*) by the successful immunization of animals and human beings with the pneumotropic streptococcal vaccine and with the virus vaccine, and (*d*) by the demonstration in filtrates of the virus [27a] and in tissue cultures of the virus [27b] of spheres, ovoids, and pairs in short and long-chain formation with the electron microscope. The Streptococcus and virus are similar or alike in tropism and antigenicity but greatly different in size. The Streptococcus appears to be the toxicogenic, antigenic phase and the virus the relatively nontoxicogenic nonantigenic, but highly invasive phase.

Additional evidence indicating the importance of the pneumotropic Streptococcus in the epidemiology, etiology, prevention, and treatment of epidemic influenza and milder forms of respiratory infection has been obtained in these studies. The isolation, filtrability, and localizing properties of the Streptococcus isolated from the nasopharynx of persons having a respiratory infection or poliomyelitis and from poliomyelitis virus (Table 1) are in accord with previous studies. Most important, immediate cutaneous reactions to intradermal injection of the respiratory streptococcal thermal antibody in persons having epidemic influenza or related respiratory infections indicating specific circulating streptococcal antigen paralleled the severity of the disease and diminished slowly during natural recovery and abruptly following therapeutic injection of thermal antibody. Reactions indicating antibody as determined on intradermal injection of antigen increased slowly during natural recovery and abruptly after therapeutic subcutaneous injection of respiratory streptococcal antibody (Charts 1, 2, 3, and 4).

The demonstrated parallelism between cutaneous reactions indicating streptococcal antigen and antibody in skin or blood of persons having respiratory infection (Table 2) and to a less degree in well persons within epidemic zones and the isolation of the pneumotropic Streptococcus from outdoor air during such epidemics are considered of great epidemiological importance (Table 3).

The results reported were obtainable only if the underlying principles as regards methods for the isolation of the specific type of Streptococcus and maintenance of specificity were fulfilled. The usual bacteriological mediums and methods did not suffice. On the basis of the results of these combined studies of the Streptococcus and the virus in influenza, the admirable studies which have been made of viruses, especially of influenza virus, lack breadth of concept as regards their source and their relationship to the bacteria, resulting in inadequate bacteriological methods.

Prophylactic immunization against epidemic influenza, as should be expected, has been successful with both the streptococcal and the viral vaccines. Protection with the streptococcal vaccine was afforded in numerous annual outbreaks by many physicians throughout 34 years. Composite pneumotropic streptococcal vaccines made from the undenatured streptococci from a central source were found to protect geographically against influenza and milder respiratory infections. Under proper dosage such streptococcal vaccines proved harmless and nonsensitizing. Recurring respiratory infections in highly susceptible persons have been prevented by prophy-

lactic immunization with composite pneumotropic streptococcal vaccines. Preliminary time-consuming and expensive studies to determine the presence and the type of virus at hand often prove fallacious, because new strains appear during the course of epidemics. These studies are not necessary to afford protection with the streptococcal vaccine. The streptococcal vaccines alone or in combination with the newly developed streptococcal thermal antibody prepared from composite suspensions have been found to be highly efficacious in treatment of epidemic influenza, of milder types of acute respiratory infection, of bronchitis, bronchial asthma, and sinusitis, that so often follow influenza and other types of respiratory infection due to or associated with the Streptococcus. Immunity to influenza induced with virus vaccines, while more enduring than with the streptococcal vaccine, has certain disadvantages. Protection should accrue, because by special methods both the specific type of Streptococcus and the filtrable "virus" phase have been demonstrated. The preparation and use of the virus vaccines are difficult and expensive. Untoward local and constitutional reactions due to primary toxicity of the vaccine prepared from chick embryo cultures of the "virus" or to sensitization to egg and chicken on repeat injections do occur. Antiviral serums suitable for treatment of influenza are nonexistent; whereas pneumotropic antistreptococcal serums and now the pneumotropic streptococcal thermal antibody have been shown to be effective in causing a prompt reduction of circulating antigen, an increase in circulating antibody, and a concomitant clinical improvement.[29b]

On the basis of long-continued studies, it is suggested that the nonhemolytic Streptococcus normally present in the nasopharynx of human beings and broadly in nature tends to acquire "pneumotropic" virulence of varying degree in temperate climates during winter months and sometimes otherwise, resulting in epidemics of influenza and milder types of respiratory infections. As such infections occur, a "virus" or filtrable phase of the "Streptococcus may develop and propagate in varying proportions during the course of the disease and epidemics. Cessation of epidemic outbreaks according to these studies would appear to be due mainly to two factors: (*a*) the acquisition of specific resistance from subclinical and clinical infection in the general population and (*b*) the seasonal loss of "pneumotropic" virulence of the Streptococcus and perhaps of the associated virus.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

OTITIS MEDIA AND COMPLICATIONS

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THE ARTICLES on otitis media and its complications which appeared in 1952 are listed under several headings. The reviewer's comments have been placed in brackets.

CHEMOTHERAPY AND BIOTHERAPY

Lewis and Hewlett [1] (London, England) treated a group of 25 children with acute otitis media by daily injections of penicillin. They treated a similar group with chlortetracycline (Aureomycin) and another with chloramphenicol (Chloromycetin). They found that it was very hard to get children to take chlortetracycline or chloramphenicol, especially those between 1 and 4 years of age. They found that in the children who took the drugs regularly the results were similar. Cultures were made and sensitivity tests were done routinely. When they found that an organism was insensitive to a drug used the drug was changed. Two mastoid operations were necessary in the entire group. In order to compare a control group, who had no treatment, a group of 23 cases were reviewed in which the patients were treated in 1944 and 1945, before penicillin was available. There were five mastoidectomies in this group. They concluded that penicillin would remain the drug of choice for acute otitis media until the newer antibiotics became cheaper and more palatable.

Nielsen [2] (Copenhagen, Denmark) treated acute suppurative otitis media in 57 cases with procaine penicillin and in 57 similar cases with Dipenicillin, a preparation containing quickly soluble sodium penicillin and slowly soluble penicillin procaine. The cases were similar as to age of the patients and types of organisms. All the pneumococci and streptococci were very sensitive to penicillin. The infections treated with Dipenicillin all cleared up much quicker than those for which penicillin procaine alone was given.

Herberts [3] (Uppsala, Sweden) found that in about 20% of his cases of otitis media organisms resistant to penicillin could be cultured and he thought that the percentage of resistant organisms was increasing. He had observed some cases in which the original penicillin-sensitive organism seemed to be destroyed and later cultures showed another type of organism, which was penicillin-resistant. [Many others have observed that the organisms now found in cases of acute otitis media and other conditions are more resistant to penicillin. Much larger doses should be given, other

1. Lewis, R. S.; Gray, J. D., and Hewlett, A. B.: Penicillin, Aureomycin, and Chloramphenicol in the Treatment of Acute Otitis Media, J. Laryng. & Otol. **66**:142-147 (March) 1952.

2. Nielsen, J. C.: Effect on Acute Suppurative Otitis Media of Procaine Penicillin Administered Once Daily Compared with the Effect of the Combination Preparation Dipenicillin, Acta oto-laryng. **42**:225-242 (June) 1952.

3. Herberts, G.: Some Aspects of the Modern Treatment of Infections, Acta oto-laryng. **42**:30-34 (April) 1952.

antibiotics may be given, or combinations of antibiotics may be given. Sensitization tests are useful but may be misleading; they are slow and rather expensive for routine use.]

Goodhill [4] (Los Angeles) in an article on current chemotherapy stressed the rapidity with which ideas change concerning the use of chemotherapy. With the rapid changes in the sensitivity of organisms and the rapid introduction of new drugs, "Our formulas are worthless the day after they are published." He urged greater respect for the toxic effects of the drugs themselves and urged that the masking effect in mastoiditis should not conceal the dangers of intracranial spread and damage to hearing. He also mentioned that the results of in vitro tests may be contradicted by the in vivo performance. "The drugs should be used to reinforce, not weaken, our older medical and surgical techniques."

Lazar, Goldin, and Auerbach [5] (Chicago) reported the use of neomycin in treating otitis media of infants and children, with especial reference to infections caused by Pseudomonas aeruginosa. They found 7 children and 12 adults with otitis media caused by Pseudomonas organisms. Neomycin was given intramuscularly and locally as ear drops in treatment of the infants and children. Adults were given local treatment only, a solution of 200 units per cubic centimeter being used. There were no bad reactions noted.

Friedmann [6] (London, England) made an extensive study of Staphylococcus aureus organisms found in cases of ear infections, with special attention to penicillin- and streptomycin-resistant strains. He concluded that penicillin is still the antibiotic of choice in the treatment of Staphylococcus infections. [This article will be commented on further under "Bacteriology."]

LOCAL USE OF ANTIBIOTICS

This year many writers have commented on the local use of antibiotics in the ear, especially for external otitis and chronic otitis media.

Rutter and Ballantyne [7] (England) treated 54 patients with chronic suppuration of the middle ear and old mastoid cavities by local antibiotics applied as powder. The antibiotics were all prepared in a sterile lactose powder, penicillin 2,000 units per gram, streptomycin 1%, chloramphenicol 25%, and oxytetracycline (Terramycin) 2% being used. Sensitization cultures were made, but the authors found that many infections cleared up quickly when medications were used to which the organisms had not appeared to be sensitive in vitro. They think the reason may be that the in vitro tests were devised to expose the germ to the amount of antibiotic which would be obtained when given systemically, while local application might give a much higher concentration.

4. Goodhill, V.: Current Chemotherapy in Ear, Nose and Throat Diseases, Ann. West. Med. & Surg. **6**:677-679 (Oct.) 1952.

5. Lazar, A. M.; Goldin, M., and Auerbach, H.: Treatment of Otitis Media Caused by Pseudomonas Aeruginosa in Infants with Neomycin, A. M. A. Arch. Otolaryng. **55**:444-450 (April) 1952.

6. Friedmann, I.: Bacteriological Studies in Otitis, J. Laryng. & Otol. **66**:175-180 (April) 1952.

7. Rutter, P. E. H., and Ballantyne, J. C.: Local Use of Antibiotics in Chronic Suppuration of the Middle Ear and Mastoid, with Particular Reference to Terramycin, Lancet **2**:314-315 (Aug. 16) 1952.

They concluded that oxytetracycline "undoubtedly gave the most satisfactory results in this series." They feel that oxytetracycline is worthy of further extensive trial. No irritative effects were mentioned.

Ausband and Harrill [8] treated 54 patients having chronic ear infections with local applications of chloramphenicol. The drug was prepared by mixing 0.5 gm. of chloramphenicol with 2 cc. of 70% alcohol. Five drops of this mixture was instilled in the ear four times daily. Eleven patients with external otitis were cured. Of 20 ears with chronic otitis media and central perforations, 19 became dry. Of three patients with a marginal perforation only one was cured. Of 15 ears with infected radical-mastoidectomy cavities, 9 became dry and 6 were markedly improved.

There were three patients in whom irritation developed, and all recovered within 48 hours after use of the drug was stopped.

Vaheri and Savolainen [9] (Helsinki, Finland) used antibiotics in the treatment of external otitis. Sensitization tests were done and were found to be of great help. The authors found Staph. aureus to be most frequently responsible in acute, subacute, and chronic external otitis. Ps. aeruginosa was second in importance. Their best results were obtained with dihydrostreptomycin, sulfathiazole, and 3% acetic acid. Penicillin was found to have no therapeutic value. Harmful side-effects "were not generally observed," but "chloromycetin [chloramphenicol] and aureomycin [chlortetracycline] may cause temporary irritation."

Magauran [10] (England) suggested that a cheap and convenient method of application of antibiotics to the middle ear could be devised, using the powder still in the original capsule. "To construct the insufflator in simplest form, the capsule is perforated at each end by a fine pin and it is then fitted into the mouth of a small rubber bulb such as is attached to a glass medicine dropper."

Tremble and Baxter [11] (Montreal) reviewed the literature on external otitis. They advised sensitization tests in cases of stubborn bacterial infections and caution in the use of antibiotics. They have seen several severe local toxic reactions to penicillin and streptomycin and are careful in their use. They state that any local application may be an irritant, and they make it a rule never to use any local application longer than five days. In cases of acute infection with much swelling of the canal they advise wicks saturated with a urea-acetic acid solution or aluminum acetate solution (Burow's solution, or 2% acetic acid and 5% aluminum acetate).

[In using antibiotics locally for ear infections for several years, I have seen only two very severe reactions. Both patients were highly allergic persons; one had had a severe general reaction to penicillin when it was used intramuscularly for another illness. One of these patients reacted to oxytetracycline used locally, and the other reacted to streptomycin. Caution should be used, especially when dealing with a patient who has an allergic tendency. For the nonallergic patient who fails to respond to simpler medications, the local use of antibiotics is usually very successful.]

8. Ausband, J. R., and Harrill, J. A.: The Local Use of Chloromycetin in Infected Ears, North Carolina M. J. **13**:568-571 (Oct.) 1952.

9. Vaheri, E., and Savolainen, T.: The Importance of Bacteriological Study in the Treatment of External Otitis, J. Laryng. & Otol. **66**:253-258 (June) 1952.

10. Magauran, W. H. B.: Local Application of Antibiotics to the Middle Ear, Lancet **2**:589-590 (Sept. 20) 1952.

11. Tremble, G. E., and Baxter, J. D.: Otitis Externa: A Controversial Subject, A. M. A. Arch. Otolaryng. **56**:241-249 (Sept.) 1952.

BACTERIOLOGY

Meurman, Kankkunen, and Savolainen [12] (Finland) reported bacteriological examinations of 85 patients with chronic otitis media with central perforations. They found the Streptococcus to be infrequent, the commonest organism being Staph. aureus, which occurred in 33% of cases. Gram-negative rods were found in 41%, the commonest being Ps. aeruginosa. Resistance tests were done during treatment, and they frequently found that resistance increased during treatment. Pseudomonas organisms rapidly became resistant to streptomycin if the antibiotic was used in inadequate doses. Although details of treatment were not given, they found streptomycin the most effective drug for most organisms and boric acid to be very effective against Ps. aeruginosa. "Treatment with one or more of the test substances, using local or systemic therapy, was successful."

Hardy and associates [13] (Florida) made cultures in 646 cases of external otitis seen at the air force bases at Tampa and Jacksonville, Fla. They also cultured material from 1,377 normal ears as controls. They found many cases of external otitis in this area, confirming many other reports that otitis externa is apt to be common in hot humid areas. Cultures of material from normal ears rarely showed Pseudomonas and other pathogens, while Pseudomonas occurred in 65% of infected ears. There were few infections caused by fungi. They performed sensitization tests on the isolated organisms and found that oxytetracycline and sulfadiazine were the most effective in vitro. There was no report on the clinical results of the different medications.

Friedmann [6] (of the Pathology Department, University of London) cultured the exudate in 1,469 cases of infections of the ear, including acute otitis media, chronic otitis media, and external otitis. He used the term Staph. aureus for the coagulase-positive organisms, regardless of the color of the colonies and their ability to hemolyze blood on culture plates.

Although 14.9% of the coagulase-positive Staph. aureus organisms were penicillin resistant, he still considered penicillin the antibiotic of choice in treating Staphylococcus infections. Of 366 penicillin-resistant strains of Staph. aureus, only 6 were resistant to streptomycin. He found that Staph. aureus was present in 46% of the 664 cases of acute otitis media, in 44% of the cases of external otitis, and in 33% of 318 cases of chronic otitis media.

Rantz [14] (Stanford University) reviewed the records of 84 cases of acute otitis occurring from 1940 to 1943. In all cases the infection was caused by the hemolytic Streptococcus. All patients were 12 years old or under. Antibiotics were not available, and the sulfonamides were not used early. The infections were severe and prolonged, and 20% of the patients required mastoidectomy. There were many complications and many recurrences of infections. This study was done as a background for comparison with later cases in which antibiotics were used.

12. Meurman, Y.; Kankkunen, S., and Savolainen, T.: Bacteriological Examinations and Determinations of Bacterial Resistance in Chronic Suppurative Otitis Media with Central Perforation, Acta otolaryng., Supp. 100, pp. 6-13, 1952.

13. Singer, D. E.; Freeman, E.; Hoffert, W. R.; Keys, R. J.; Mitchell, R. B., and Hardy, A. V.: Otitis Externa: Bacteriological and Mycological Studies, Ann. Otol. Rhin. & Laryng. **61**:317-330 (June) 1952.

14. Rantz, L. A.: Hemolytic Streptococcus Otitis Media, Stanford M. Bull. **10**:32-38 (Feb.) 1952.

Siirala and Lahikainen [15] (Turku, Finland) took samples of exudate from the middle ear of patients with otitis media and tested the bacteriostatic effect on numerous bacteria. The exudate was aspirated through the intact drum while the patient was under general anesthesia. One hundred twenty-six samples were taken. Similar tests were run on material aspirated from the maxillary sinus in 15 cases. There was definite inhibition of bacterial growth of some organisms by 53% of the samples from the middle ear and by 56% from the maxillary sinus. Blood serum was taken from 18 of these patients and was found to be less effective. Dilution of the serum destroyed its effectiveness. Drying the exudate and storing it for three weeks did not destroy its bacteriostatic power. "This effect seems to resemble the lysozyme effect described by Fleming."

Goodale [16] (Boston) has been observing the effect of autogenous vaccine used for chronic infections of the upper respiratory tract. The organism he finds most important in these chronic infections is the hemolytic coagulase-positive Staph. aureus. After long observation he has become convinced that the majority of patients (possibly 75%) were helped. In the discussion of Goodale's article Clerf stated that he considered the use of autogenous vaccines for chronic infections of the lower respiratory tract very valuable. He agreed with Goodale that small doses were important.

CHRONIC OTITIS MEDIA

Simonton [17] (the Mayo Clinic) gives his method of selection for surgical procedures of patients with chronic suppurative otitis media. It is based on the classification of Lillie published in 1936. Group 1 comprises patients with an odorless mucoid discharge. Perforations may be of any size or location but are usually in the anteroinferior quadrant. Group 2 includes patients with evidence of disease in the middle ear, such as polypi, granulations, or necrosis of the ossicles. Group 3 includes patients with defects in the attic. Small perforations in Shrapnell's membrane or above are worse than large perforations. The discharge is purulent and fetid. X-rays may show the sclerotic mastoid found in the previous groups and sometimes areas of decreased density. Group 4 includes patients with headache, pain, vertigo, nausea, fever, facial palsy, and increased intracranial pressure.

Treatment usually cures patients in the first two groups and may cure those in the third group, but the fourth group should have surgery at once. Surgical management is thoroughly discussed. [This is an excellent summary of the subject.]

Boies and Younger [18] (Minneapolis) reviewed the different factors that may cause absence of pneumatization in the temporal bone and quoted Schwartz's recent paper on the hereditary factor. They took x-rays of 300 children and young adults and found about the same percentage of nonpneumatized mastoids as previous writers, 15. They think that with the poor pneumatization there is a hyperplastic type

15. Siirala, U., and Lahikainen, E. A.: Some Observations on the Bacteriostatic Effect of the Exudate in Otitis Media, Acta. otolaryng., Supp. 100, pp. 20-25, 1952.

16. Goodale, R. L. and Mangiaracine, A. B.: Clinical Observations on the Use of Autogenous Vaccine Made from Cultures of Coagulase Positive Hemolytic Staphylococcus Aureus, Tr. Am. Laryng. Rhin. & Otol. Soc., pp. 22-37, 1953.

17. Simonton, K. M.: Selection of Patients for Surgical Procedures in Chronic Suppurative Otitis Media, J. A. M. A. **150**:461-464 (Oct. 4) 1952.

18. Boies, L. R., and Younger, L. I.: Abnormal Pneumatization of the Temporal Bone, Ann. Otol. Rhin. & Laryng. **61**:836-849 (Sept.) 1952.

of mucous membrane of the middle ear and mastoid antrum instead of the thin mucoperiosteum found in well-pneumatized mastoids. They believe that absence of pneumatization, and the hyperplastic mucous membrane, tend to promote chronicity of serous otitis and otitis media.

[In the discussion Meltzer quoted Cheatle, who held that the compact mastoid was normal for certain persons. He doubted that fluid accumulation was due to abnormal pneumatization and accompanying thickening of the mucosa.]

MENINGITIS

Gibson and James [19] report on the treatment of 31 patients with pneumococcal meningitis at the Presbyterian Hospital, New York City, from 1944 to 1950. With the exclusion of three patients who died within one day after admission (one with severe diabetes and another with multiple skull fractures), the recovery rate was 100%.

The authors urge intrathecal administration of penicillin, since systemic use alone has not produced nearly as high a percentage of cures and since intrathecal administration of penicillin produced no irritative symptoms in this series. They advise an intrathecal dosage of 25,000 units dissolved in 10 cc. of isotonic sodium chloride solution and an intramuscular dosage of 2,000,000 to 4,000,000 units given daily for two weeks.

They quoted Lepper and Dowling, who reported a mortality rate of 30% in a penicillin-treated group and of 79% in a group treated with penicillin and chlortetracycline and suggest that this combination may be contraindicated.

Feibush, Murphy, and Lubart [20] analyzed results in 22 cases of pneumococcal meningitis and, among other conclusions, decided that intrathecal use of penicillin was not an essential part of treatment and quoted others who had come to the same conclusion. Their mortality rate was 64%. In one-third of their cases meningitis developed after "upper respiratory infections and (or) otitic infections."

Bunn and Peabody [21] reported 20 cases of pneumococcal meningitis, in 5 of which the meningitis developed after otitis media. The patients were given 1,000,000 units of aqueous crystalline penicillin every two hours. No penicillin was used intrathecally "because of its irritating qualities and poor penetration." Seventy-five per cent were cured.

Ginsberg and Hyman [22] report a case of meningitis caused by Ps. aeruginosa. The patient was a 64-year-old woman who had a radical mastoidectomy done because of a carcinoma of the external auditory canal. A cure was obtained by the combined

19. Gibson, C. D., and James, D. G.: Treatment of Pneumococcal Meningitis Since the Introduction of Penicillin, Lancet **2**:1203-1205 (Dec. 20) 1952.

20. Feibush, J. S.; Murphy, E. J., and Lubart, A.: Pneumococcal Meningitis in Adults, Ann. Int. Med. **37**:65-74 (July) 1952.

21. Bunn, P. A., and Peabody, G.: Treatment of Pneumococcal Meningitis with Large Doses of Penicillin: Series of 20 Consecutive Cases, A. M. A. Arch. Int. Med. **89**:736-742 (May) 1952.

22. Ginsberg, I. A., and Hyman, G. A.: Combined Aureomycin and Streptomycin Therapy of Pseudomonas Aeruginosa (Bacillus Pyocyaneus) Meningitis, with a Case Report, Ann. Int. Med. **37**:194-197 (July) 1952.

use of chlortetracycline, streptomycin, and penicillin. Streptomycin was given intrathecally in 100 mg. doses daily for three days as well as 0.5 gm. every six hours intramuscularly. Sensitivity tests were not done.

LATERAL SINUS THROMBOSIS AND OTITIC HYDROCEPHALUS

Steegmann and Williamson [23] reported an interesting case of a 35-year-old man in whom brain abscess developed from a chronic otitis media dating from childhood. Drainage of the temporal lobe was done, and the cerebral symptoms improved. Radical mastoidectomy was done, and a thrombosed lateral sinus was found. In spite of the fact that the patient continued to have a high fever, the internal jugular vein was not ligated. Ten days later the patient died, and autopsy showed a large pulmonary embolus in the main pulmonary artery extending into the inferior branches.

[This case represents a valuable lesson. One must not neglect ligation of the internal jugular vein when the patient continues to have a septic temperature after mastoidectomy has demonstrated a lateral sinus thrombosis.]

Gabriel [24] (London, England) reported the interesting case of a boy of 16 who had a draining left ear for three years. Operation showed a thrombosed lateral sinus, which was treated by packing (without ligation of the jugular vein) and use of penicillin with "sulfonamide." The patient improved, but at the end of a week paralysis of the sixth nerve on the opposite side and bilateral papilledema developed. Lumbar puncture showed a clear fluid with a pressure well over 300 mm. A diagnosis of otitic hydrocephalus was made, and he was treated by daily lumbar puncture for six days, after which the pressure came down to 180 mm. Two more punctures at 48-hour intervals showed normal pressures. There was an incomplete facial paralysis on admission. The patient recovered, with no paralysis and with normal hearing.

In an editorial in the *Lancet* [25] it is stated that the general opinion as to the cause of otitic hydrocephalus is thrombosis of the intracranial sinuses. The raised intracranial pressure may be due to either of two mechanisms. The clot may spread from one lateral sinus to the torcular and in this way shut off the opposite lateral sinus and thus obstruct the main venous return from the cranial cavity, or a clot in the superior longitudinal sinus may block the arachnoid villi and in this way impede the absorption of cerebrospinal fluid. This explanation is chiefly based on work of Symonds.

BRAIN ABSCESS

Shuster [26] (Philadelphia) reminds us that the otolaryngologist is almost always first consulted in cases in which brain abscess is apt to develop as a complication of infection in the nose and ear and that he is duty-bound to be alert to the symptoms of such infection. He states that the most striking symptom of brain abscess is the listless, disinterested mental attitude of the patient. The characteristic type of headache is not severe but is dull and thumping and sufficient to keep the patient awake

23. Steegmann, A. T., and Williamson, W. P.: Clinical Pathological Conference, Neurology **1**:483-488 (Nov.-Dec.) 1951.

24. Gabriel, C. E.: A Case of Lateral Sinus Thrombosis Complicated by Otitic Hydrocephalus with Seventh Nerve and Contralateral Sixth Nerve Paralyses, J. Laryng. & Otol. **66**:290-291 (June) 1952.

25. Intracranial Thrombosis, Editorial, Lancet **2**:174-175 (July 26) 1952.

26. Shuster, B. H.: Brain Abscess: Otogenic and Rhinogenic, A. M. A. Arch. Otolaryng. **56**:114-120 (Aug.) 1952.

in spite of his drowsiness. Macewen's sign should be looked for: a pulse slower than would be expected with the degree of temperature. This is not persistent but recurs occasionally.

Localizing symptoms and signs are reviewed and then the problem of the approach to the drainage of the area and the method of drainage. He states that the abscess should be drained as soon as the diagnosis can be made and through the nearest area to the surface. This area is usually nearest the focus of infection. He advises drainage through a small rubber tube after location of the abscess with a cannula or grooved director. [This excellent article should be read in the original.]

Wood [27] (Newcastle, England) reports his results in the treatment of 41 patients with brain abscess. With the elimination of three patients moribund on admission, the mortality was only 14.3%. Treatment was based on repeated aspiration and injection of penicillin through suitable burr holes. Early evacuation was urged. This conservative treatment usually resulted in shrinking of the abscess to a small harmless scar. In a few cases it failed to shrink and required excision. The process of shrinking was observed by means of injected thorium dioxide (Thorotrast).

LABYRINTHITIS

Watson [28] (England) has decided after 27 years of practice and observation of more than 3,000 cases of mastoid operations that the "mutilating operation of translabyrinthine drainage" is quite unnecessary and should not be performed. He was taught by Fraser in 1920 to perform caloric tests in cases of mastoiditis. Within three years he had three patients with labyrinthitis on whom he operated. All three patients died. He has seen patients with mastoiditis with severe labyrinthine symptoms recover with no loss of hearing. He concluded that they had serous (not suppurative) labyrinthitis and that it was impossible to distinguish between serous and suppurative labyrinthitis by the symptoms. After stopping the performance of labyrinthine drainage operations he saw only one death from labyrinthitis and meningitis.

Archer [29] in his presidential address to the Otology Section of the Royal Society of Medicine reviewed his experience of 30 years with labyrinthitis. In a series of 1,379 patients who underwent mastoid operations there were 52 on whom labyrinth operations were done. There were 12 deaths. He divided his cases into three groups: (1) those occurring from 1921 to 1936, (2) those observed between 1937 and 1945 (when sulfonamides were available), and (3) those occurring from 1946 to 1951 (when there was penicillin). There were 7 deaths among 15 cases in the first period, 4 deaths among 24 cases in the second group, and 1 death among 9 cases in the last group. His conclusions were as follows: 1. In meningitis occurring with acute otitis media heavy chemotherapy should be carried out and simple mastoidectomy should be done. 2. In cases of chronic otitis media in which severe labyrinthine symptoms occur a labyrinthotomy is justifiable if symptoms do not yield to simpler treatment. 3. With a necrotic labyrinth capsule and a dead labyrinth, a labyrinthotomy is

27. Wood, P. H.: The Treatment of Brain Abscess, J. Laryng. & Otol. **66**:71-83 (Feb.) 1952.

28. Watson, D.: Labyrinthitis, J. Laryng. & Otol. **66**:247-252 (June) 1952.

29. Archer, G. E.: Suppurative Labyrinthitis: President's Address, Proc. Roy. Soc. Med. **45**:121-127 (March) 1952.

imperative. 4. In some cases of recurring serous labyrinthitis it is wise to perform labyrinthotomy between attacks rather than to wait until a purulent labyrinthitis develops.

CHOLESTEATOMA

Ojala and Saxén [30] (Helsinki, Finland) made careful histological studies on 30 temporal bones to determine the cause of attic cholesteatoma. These specimens were obtained from autopsies of persons over 50 years of age. The authors concluded that cholesteatoma may arise from Shrapnell's membrane even in the absence of a perforation. They think that during an infection the normal correlation between epidermis and mucosa is destroyed and that the epidermis may project atypical processes through Shrapnell's membrane. An additional prerequisite is that the mucous membrane remains hyperplastic after cessation of the inflammation and does not become completely fibrous. The most susceptible persons are those with constitutionally inferior mucous membrane that has been subject to mild chronic infections in childhood.

Bauer [31] reported two postoperative mastoid cases in which there developed cholesteatomatous masses just below the skin. In the first case there were two separate "cysts," one the size of a cherry and the other the size of an egg. There was no communication between the cysts, and the mastoid cavity was clean. The cysts were filled with cholesteatomatous material. In the second case a cyst the size of a walnut developed at the lower edge of the mastoidectomy scar.

Peterson [32] (Rye, N. Y.) reported 11 cases of cholesteatoma formation occurring in a series of 52 cases of radical mastoidectomy operations in which primary skin grafts had been done. The cholesteatoma formations were extensive; masses of epithelium and granulations filled the operative cavity completely. Recurrence came on a year or two after operation in cavities that had previously been clean and dry.

Before 1946 he had done 268 radical mastoidectomy operations and noticed no similar reactions. Since the 52 operations performed since 1946 had been done more thoroughly and carefully, the endaural technique being used, he concluded that the granulations and cholesteatoma formation were due to the skin grafts. He quotes Begley and Williams, who stated that "in certain instances it has seemed that the eventual result is better when skin grafting is not used, because of occasional tendencies in the grafted skin for an eczematoid reaction to develop." Peterson is disturbed that other surgeons have not reported similar cases. [If others confirm these observations the advisability of skin grafts is questionable.]

EXTERNAL OTITIS

Senturia and Marcus [33] (St. Louis) presented a concise classification, description, and differential diagnosis of all varieties of lesions of the external ear. Therapy of the different conditions was not discussed.

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30. Ojala, I., and Saxén, A.: Pathogenesis of Middle-Ear Cholesteatoma Arising from Shrapnell's Membrane (Attic Cholesteatoma), Acta oto-laryng., Supp. 100, pp. 33-54, 1952.

31. Bauer, F.: Implantation Dermoid in a Mastoidectomy Scar as a Manifestation of Cholesteatosis, J. Laryng. & Otol. **66**:617-619 (Dec.) 1952.

32. Peterson, N. V.: Cholesteatoma in Radical Mastoidectomy Cavity Secondary to Use of Primary Skin Grafts, A. M. A. Arch. Otolaryng. **56**:49-56 (July) 1952.

33. Senturia, B. H., and Marcus, M. D.: Etiologic Classification of Diseases Involving the External Ear, Ann. Otol. Rhin. & Laryng. **61**:18-32 (March) 1952.

Lett [34] (Florida) reported 100 cases of external otitis in which 5% 4-amino-2-methyl benzene sulfonamide hydrochloride (Sulfamylon) in methylcellulose was used. After sensitivity tests on many organisms cultured from material from ears with external otitis (chiefly Ps. aeruginosa) it was decided to try this compound, which had in vitro shown more inhibitory effect than the antibiotics. The canals were cleaned with hydrogen peroxide, dried, and packed with cotton wicks saturated with the drug. The patient was given a bottle of the solution and told to moisten the wick twice daily. After the acute stage passed, 2% acetic acid or boiled vinegar was substituted. Only two patients showed reactions to the drug.

Vaheri and Savolainen [35] (Finland) cultured material from 140 infected ears found to have external otitis. Sensitization tests were done in all cases. These were seen within one year. It was surprising that among 80 ears of the 140 there were pure cultures of Staph. aureus in 50 cases and of Pseudomonas in 20. The different strains of Pseudomonas proved sensitive only to streptomycin, sulfathiazole, and boric acid. "The resistance test was a valuable guide in the choice of treatment." Results of treatment were not given.

SECRETORY OTITIS MEDIA

Suehs [36] (Austin, Texas) wrote a comprehensive article on secretory otitis, reviewing the literature carefully and giving an analysis of 342 cases seen by himself in five years. His table of incidence of serous otitis in various localities is interesting and proves that high altitude and low humidity are important factors in prevention of this condition. (Statistics were taken from articles and from letters from representative otologists over the country.) Even though he is sure that in some of his cases the condition was due to allergy, he found only one eosinophile in examination of 50 slides made of secretions from the aspirated fluid. He reported two cases in which the condition was due to "perhaps autonomic dysfunction and arteriolar spasm" and responded quickly to vasodilator drugs, in one case to histamine and in the other to nicotinic acid, after many incisions and other treatment had failed.

Singleton [37] (Dallas, Texas) states that the diagnosis of otitis media with effusion can usually be made from the history alone. The patient complains of painless hearing impairment, which may vary with changing the position of the head. The patient feels the fluid move about in the ear if the middle ear is partly filled. In cases of stubborn infection a careful search must be made for underlying factors.

AEROTITIS

Hyde [38] (Randolph Field, Texas) reviewed 93 articles on aerotitis. He agreed with many observers that the danger of suppurative otitis media developing during flying if the flier is suffering from acute respiratory infections is not great but that optional flying should be postponed when possible. The majority of writers felt that excess lymphoid tissue was not often a cause of obstruction in adults.

34. Lett, J. E.: A Therapeutic Procedure for External Otitis, Air Force School of Aviation Medicine, Randolph Field, Texas, Report No. 1, Project No. 21-32-026, Aug., 1952.

35. Vaheri, E., and Savolainen, T.: Bacteriological Examinations and Determinations of Bacterial Resistance in External Otitis, Acta oto-laryng., Supp. 100, pp. 14-19, 1952.

36. Suehs, O. W.: Secretory Otitis Media, Laryngoscope **62**:998-1027 (Sept.) 1952.

37. Singleton, J. D.: Otitis Media with Effusion, South. M. J. **45**:798-801 (Sept.) 1952.

38. Hyde, R. W.: Aerotitis Media: A Critical Review, Ann. Otol. Rhin. & Laryng. **61**:937-975 (Dec.) 1952.

TECHNIQUE

Gutteridge [39] describes a new horizontal incision he has been using for simple and radical mastoidectomies. The incision extends straight backward from the anterior attachment of the concha for 2 in. (5 cm.). The advantages claimed are the invisibility of the scar and the absence of change in position of the ear.

[It seems unlikely that this incision could give as adequate exposure as the Lempert incision.]

Torok [40] reports that for years he has been using mild negative pressure applied to the external ear canal in cases of "negative balance disorders" of the middle ear. Aspiration is done with the Siegle speculum or, preferably, Bruning's speculum. It is done alone or before inflation of the Eustachian tube. In some cases aspiration is more effective than tubal inflation. Tinnitus due to blocking of the Eustachian tube decreases at once on application of suction.

Withers, Dickson, and Wattleworth [41] (Houston, Texas) used thin split-thickness skin grafts in 26 cases of radical mastoidectomies and observed as controls 20 cases in which grafts were not used. In the 26 cases in which grafts were used there was healing with dry cavities in an average time of 9.3 weeks, while in the controls healing took an average of 25.6 weeks. They made a mould of a piece of rayon and gauze saturated with petrolatum and paraffin. The graft was placed over this mould and left undisturbed for 10 to 12 days. They found that thin grafts took better than thick and that grafts worked very well over exposed facial nerve, dura, or lateral sinus. They decided that immediate grafting should be done in all cases.

Adin [42] used preserved cancellous bone grafts to fill the opening of the Eustachian tube in five patients undergoing radical mastoidectomy. In three patients the tube remained closed and the middle ear remained dry. One patient had persistent discharge from the middle ear and a patent Eustachian tube. The fifth patient did not return for postoperative observation. Autogenous bone from the iliac crest was suggested as being more valuable; another series is being treated with grafts of such bone.

[The advisability of trying to close the Eustachian tube has been questioned by good authorities.]

MISCELLANEOUS

Dingley [43] (England) reported a case of eosinophilic granuloma involving both middle ears and mastoids in a boy of 2. Both ears had drained for six weeks, and the canals were choked with pale granulations. The diagnosis was made when x-rays showed translucent areas beyond and above the mastoids. X-ray treatment was given, and the mastoids were cleaned out by operation. At the time of the report the child was improving but did not seem to hear.

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39. Gutteridge, E.: Mastoidectomy: A New Supra-Auricular Approach to the Middle Ear, A. M. A. Arch. Otolaryng. **55**:163-171 (Feb.) 1952.

40. Torok, N.: Effects of Aspiration in the External Ear Canal, A. M. A. Arch. Otolaryng. **55**:157-162 (Feb.) 1952.

41. Withers, B. T.; Dickson, J. C., and Wattleworth, K. L.: Primary Split Thickness Skin Grafting of Radical Mastoid Cavities, Ann. Otol. Rhin. & Laryng. **61**:656-662 (Sept.) 1952.

42. Adin, L. E.: The Use of Cancellous Bone Grafting at Tympanomastoidectomy for Permanent Eustachian Tube Closure: A Preliminary Report, Laryngoscope **62**:173-193 (Feb.) 1952.

43. Dingley, A. R.: Eosinophil Granuloma of the Temporal Bone, J. Laryng. & Otol. **66**:285-287 (June) 1952.

Harris and Spencer [44] (Cleveland) reported a case of involvement of both mastoids by eosinophilic granuloma in a child of 3. X-rays showed destruction of tissue in both mastoids. There was a discharging postural fistula on one side. The associated infection and swelling of the ear canals, as well as the postauricular sinus tract, responded well to x-ray therapy. Only three similar cases, in which both temporal bones were involved, had been reported.

Derlacki [45] (Chicago) quotes the work of Koch and Dohlman on allergic otitis media and reports several cases of external otitis and chronic otitis media which were cleared up by allergic therapy. In one case of crusty weeping external otitis of many years' duration, the condition was cleared up after an elimination diet established pork as the cause. Three recurrences were traced to foods prepared with lard and to frankfurters containing pork. One case of serous otitis cleared up with house dust therapy. Serum from the ear had shown a few eosinophiles.

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44. Harris, H. E., and Spencer, D. M.: Eosinophilic Granuloma, Bilateral Mastoid Involvement: A Case Report, Cleveland Clin. Quart. **19**:87-89 (April) 1952.

45. Derlacki, E. L.: Aural Manifestations of Allergy, Ann. Otol. Rhin. & Laryng. **61**:179-188 (March) 1952.

Case Reports

EXTRACTION OF FERROMAGNETIC FOREIGN BODIES WITH THE ALNICO MAGNET

HOWARD R. BANCKS, M.D.
JOHN J. MASTERSON, M.D.
AND
SIDNEY S. LERNER, M.D.
BROOKLYN

BY COMBINING aluminum, nickel, and cobalt with iron, physicists have created the Alnico magnet which has a long life and a much greater attractive power than the old type of permanent magnet. Its great coercive force and relatively small size make it adaptable for use in the limited spaces of the air and food passages, and it has become a worth-while addition to the armamentarium of the otolaryngologist. Although it is not an instrument for routine use, the otolaryngologist will be happy to have one available when an exigency arises, as the following case illustrates.

REPORT OF A CASE

Julia P., age 3 years, was admitted to the Norwegian Hospital, Brooklyn, with a history of having swallowed a 2¼ in. (5.72 cm.) finishing nail one week previously. The patient had no complaints, but she was admitted for observation after the nail failed to appear in the stools. X-ray studies revealed the presence of the foreign body in the stomach. With the diagnosis established, the patient was discharged for an additional week's observation at home to see whether the foreign body might pass through spontaneously.

At the end of the second week, the patient returned to the hospital and fluoroscopy revealed that the nail had not moved from its abode in the stomach. Accordingly, the child was "mummified" on the x-ray table and extraction was accomplished under fluoroscopic guidance without anesthesia. The hypopharynx was exposed with a laryngoscope, and an Alnico magnet attached to a gastric tube as designed by Equen [1] was introduced. The child was requested to swallow as the tube was gently fed down into the stomach.

The magnetic tip could be observed through the fluoroscopic screen as it approached the nail in the stomach. A distinct click was heard as the nail "jumped" to the magnet. The nail became attached to the magnet for somewhat less than half of its length, with the point trailing. After withdrawal of the gastric tube, the fluoroscope was turned off and room lights turned on. We found that we had extracted the magnet without the nail. The fluoroscope revealed that the nail was back in the stomach. On reaching the cricopharyngeus, the nail had been stripped off. However, the magnet was readily reintroduced, and this time the nail became attached for about three-quarters of its length, with the head end trailing. As the tube was gently withdrawn, the nail appeared in the pharynx, where it was grasped by the operator and extracted. Next morning, the child was discharged.

The major disadvantage of the permanent magnet lies in the inability of the operator to readily control the position of the foreign body or its presenting part

1. Equen, M.: A New Magnet for the Removal of Foreign Bodies from the Food and Air Passages, Ann. Otol. Rhin. & Laryng. **53**:775-776, 1944.

once it becomes attached. This difficulty has been overcome by the use of an ingenious electromagnetic device designed by Penta.[2]

In considering the general problem of foreign bodies in the gastrointestinal tract, we are amazed by the great variety in the size and shape of objects that may travel uneventfully through the alimentary circuit with no more treatment than patient observation and a normal diet. There are, however, instances in which

Nail lodged in the stomach.

foreign bodies which appeared relatively innocuous have, in fact, pierced the bowel.[3] We are thus in a position with our procedure to save the patient from a laparotomy.

2. Penta, A. Q.: Electro-Magnet for the Removal of Magnetic Objects from the Bronchial and Esophageal-Gastric Tract, read before the American Broncho-Esophagological Association, Atlantic City, N. J., April 7, 1948.

3. Ward-McQuaid, J. N.: Perforation of the Intestine by Swallowed Foreign Bodies: With a Report of 2 Cases of Perforation by Rabbit Bones, Brit. J. Surg. **39**:349-351, 1952. McKechnie, W. R.: Perforation of the Intestine by an Ingested Sharp Foreign Body, Australian & New Zealand J. Surg. **13**:265-266, 1944.

This case is reported to reiterate how readily magnetizable foreign bodies can be removed from the stomach. Others even have recently demonstrated the facility with which objects that have passed beyond the pylorus can be retrieved by using the Alnico magnet attached to an intestinal tube.[4]

The more conventional method of removal with forceps suffices for most foreign bodies in the air passages. However, in cases in which the object has been aspirated into the finer divisions of the bronchial tree, beyond the reach of the forceps, the magnet may be used either at the end of a rigid rod as designated by Hollinger or with the vertebrated type of rod described by Jackson.

SUMMARY

Another case report of extraction of a ferromagnetic foreign body from the gastrointestinal tract with the Alnico magnet is added to the literature. The relative ease with which this procedure is carried out is emphasized and weighed against the possibility of perforation when a course of masterful inactivity is pursued. The feasibility of this method when the object has passed beyond the pylorus has recently been demonstrated. A possible laparotomy may be obviated by this modality, which should be included in the otolaryngologist's armamentarium.

4. Whalen, E. J.: Magnet Extraction of Foreign Bodies, Ann. Oto. Rhin. & Laryng. **59**:717-725, 1950. Dorenbusch, A. A.: Ferromagnetic Foreign Bodies Beyond the Pylorus: A Report of 2 Cases in Which Successful Peroral Removal Was Accomplished by Use of the Alnico Magnet, A. M. A. Arch. Otolaryng. **55**:589-596, 1952.

CORRECTION

In the article "Carcinoma of the Middle Ear and Mastoid," by Drs. J. William Wahl and Maurice T. Gromet, in the August, 1953, issue of the Archives, the final sentence on page 125 should read: "The cavity was packed with paraffin gauze and a mastoid external dressing applied."

Clinical Notes, New Instruments and Techniques

SIMPLIFIED SYSTEM FOR LYNCH SUSPENSION LARYNGOSCOPY

ROBERT B. LEWY, M.D.
CHICAGO

The weight, complexities, and expense of the suspension gallows and its table clamp have long presented a technical, psychological, and man-power difficulty.[1] This has resulted in making use of the Lynch suspension apparatus more rare and has tended to confine its use to the larger institutions where a team of trained personnel has been developed to handle daily, weekly, or frequent cases. The situation has been hard on the occasional operator who, though well enough qualified to do this type of work, lacked the equipment or the team necessary to perform smoothly. This new device is therefore offered to help solve this problem.[2]

Since the development of the leverage principle in laryngoscopy,[3] the leverage principle has made the exposure of the anterior commissure much easier. Such exposure now depends upon the power of the fulcrum and leverage of a worm and sprocket gear system rather than upon the strength of the operator's wrists and biceps. Consequently, the need for the Lynch instrument for such purposes has diminished. However, there are still both situations and operators that require a freedom of access to the larynx which is not constricted by a tube. The Lynch apparatus offers this, and the standard laryngoscopes do not.

Thus, for a problem of large endolaryngeal tumors, or perhaps for arytenoidectomy or cordectomy in which the operator requires a wide range of motion, the Lynch instrument still has its place.

A long tongue blade is illustrated. This has frequently been found useful to engage the tip of the epiglottis and to help compensate for the back-and-forth motion that was present in the standard suspension apparatus.

From the Department of Otolaryngology, University of Illinois College of Medicine.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study, and do not necessarily reflect the opinion or policies of the Veterans Administration.

1. Le Jeune, F. E.: History of Suspension Laryngoscopy and Bronchoscopy, Doctor **17**:83 (July) 1939.

2. Lewy, R. B.: A Gear-Power Detachable Laryngoscope Holder and Simplified Position for Direct Laryngoscopy, A. M. A. Arch. Otolaryng. **58**:444 (Oct.) 1953.

3. Roberts, S. E.: A Self Retaining Dual Distal Lighted Laryngoscope with Screw Driven Fulcrum Lift, Laryngoscope **62**:215 (Feb.) 1952.

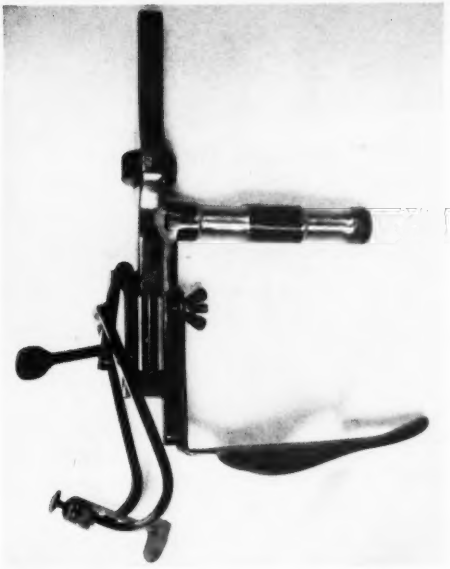

Fig. 1.—Modified Lynch apparatus with its horizontal bar which fits in the leverage laryngoscope holder.

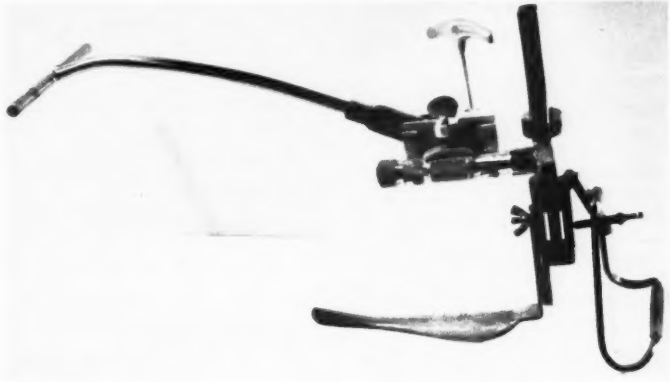

Fig. 2.—Leverage laryngoscope holder holding the modified Lynch apparatus.

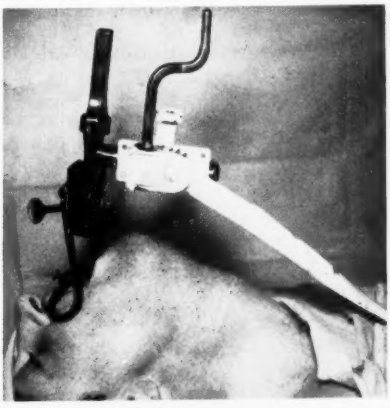

Fig. 3.—Modified Lynch apparatus being held by the leverage laryngoscope holder in the patient. Side view.

Special Reports

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

AS OF NOV. 1, 1953

Acousticon Models A-17 & A-185
Mfr., Dictograph Products, Inc.
95-25 149th St.
Jamaica 1, N. Y.

Audiotone Models 11 & 15
Mfr., Audio Company of America
5305 N. 6th St.
Phoenix, Ariz.

Audivox Models Super 67 & 70
Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Aurex Models L & M
Mfr., Aurex Corporation
117 N. Franklin St.
Chicago 10

Beltone Mono-Pac Model "Lyric"

Beltone Mono-Pac Model M

Beltone Mono-Pac Model "Rhapsody"

Beltone Symphonette Model
Mfr., Beltone Hearing Aid Co.
2900 W. 36th St.
Chicago 32

Cleartone Models 500 & 700

Cleartone Regency Model
Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Model D-1

Dahlberg Junior Model D-2

Dahlberg Model D-3 (Tru-Sonic)

Dahlberg Model D-4 (Tru-Sonic)
Mfr., The Dahlberg Company
Golden Valley
Minneapolis 22

Fortiphone Models 19LR, 20A, 21C, & 22
Mfr., Fortiphone Limited
Fortiphone House
247 Regent St.
London, W.1, England

Distr., Anton Heilman
75 Madison Ave.
New York 17

Gem Models V-35 & V-60
Mfr., The Gem Ear Phone Co., Inc.
50 W. 29th St.
New York 1

Goldentone Models 25, 69, & 97
Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Goldentone Corporation
708 W. 40th St.
Minneapolis 8

Maico Maxitone

Maico Model J

Maico Quiet Ear Models G & H

Maico Top Secret Model L

Maico UE Atomeer
Mfr., The Maico Company, Inc.
21 N. 3rd St.
Minneapolis 1

Micronic Model 303

Micronic Model Mercury

Micronic Star Model
Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Microtone Classic Model T-9

Microtone Models T-10, T-612, & 45
Mfr., The Microtone Company
Ford Parkway on the Mississippi
St. Paul 1

National Model D (Duplex)

National Ultrathin Model 504

National Vanity Model 506
Mfr., National Hearing Aid Laboratories
106 S. 7th St.
Philadelphia 6

Normatone Model C

Normatone Model D-53
Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Normatone Hearing Aid Co.
22 E. 7th St.
St. Paul 1

Otarion Models B-15 & B-30

Otarion Models F-1, F-2, & F-3

Otarion Models G-2 & G-3

Otarion Model H-1, Custom "5"
Mfr., Otarion, Inc.
4757 N. Ravenswood Ave.
Chicago 40

Paravox Model D (Top-Twin-Tone)

Paravox Model J (Tiny-Myte)

Paravox Model Y (YM, YC, & YC-7)
(Veri-Small)
Mfr., Paravox, Inc.
2056 E. 4th St.
Cleveland 15

Radiocar All-Magnetic Model 55

Radiocar Permo-Magnetic (Multipower)

Radiocar Model 62 Starlet

Radiocar Model 72

Radiocar Model 82 (Zephyr)
Mfr., E. A. Myers & Sons, Inc.
306 Beverly Rd.
Mount Lebanon
Pittsburgh 16

Distr., Radiocar Corporation
306 Beverly Rd., Mount Lebanon
Pittsburgh 16

Silvertone Models J-92 & P-15
Mfr., Johnston Hearing Aid Mfg. Company
708 W. 40th St.
Minneapolis 8

Distr., Sears, Roebuck & Co.
925 S. Homan Ave., Chicago 7

Solo-Pak Model 99
Mfr., Solo-Pak Electronics Corp.
Linden St.
Reading, Mass.

Sonotone Models 900, 910, 920, 925, 940, 966, 977, & 988
Mfr., Sonotone Corporation
Elmsford, N. Y.

Televox Model E
Mfr., Televox Mfg. Company
1307 Sansom St.
Philadelphia 7

Telex Models 99, 200, 330-B, 400, 500, 952, 953, & 1700
Mfr., Telex, Inc.
Telex Park
St. Paul 1

Tonamic Model 50
Mfr., Tonamic, Inc.
12 Russell St.
Everett 49, Mass.

Tonemaster Cameo Model
Mfr., Tonemaster, Inc.
400 S. Washington St.
Peoria 2, Ill.

Unex Midget Models 95 & 110

Unex Models 200 & 230
Mfr., Nichols & Clark
Hawthorne, Mass.

Vacolite Models J & J-2
Mfr., Vacolite Company
3003 N. Henderson St.
Dallas 6, Texas

Zenith Miniature 75

Zenith Model Royal

Zenith Model Super-Royal

Zenith "Regent"
Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

Transistor Hearing Aids Accepted

Maico Transist-Ear, Model O Mfr., The Maico Company, Inc. 21 N. 3rd St. Minneapolis 1	3 transistors and 1 battery
Otarion Model C-15 Mfr., Otarion, Inc. 4757 N. Ravenswood Ave. Chicago 40	1 transistor, 2 tubes, and 2 batteries (A & B)
Sonotone Model 1010 Mfr., Sonotone Corporation Elmsford, N. Y.	1 transistor, 2 tubes, and 2 batteries (A & B)
Telex Model 954 Mfr., Telex, Inc. Telex Park St. Paul 1	1 transistor, 2 tubes, and 2 batteries (A & B)
Zenith Model Royal-T Mfr., Zenith Radio Corporation 5801 W. Dickens Ave. Chicago 39	3 transistors and 1 battery

Semiportable Hearing Aids

Ambco Hearing Amplifier (Table Model)
Mfr., A. M. Brooks Company
1222 W. Washington Blvd.
Los Angeles 7

Aurex Semi-Portable
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Precision Table Hearing Aid
Mfr., Precision Hearing Aids
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50
Mfr., Sonotone Corporation
Elmsford, N. Y.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society

(Société Canadienne d'Otolaryngologie)

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Minaki Lodge, Minaki, Ont.

Fifth International Congress of Oto-Rhino-Laryngo-Broncho-Esophagology

Place: Amsterdam, Netherlands.
President: Prof. Dr. Eelco Huizinga.
General Secretary: W. H. Struben, Viottastraat 1, Amsterdam-Z.
Scientific Secretaries: Dr. J. Bijtel and Dr. P. G. Gerlings.

International Bronchoesophagological Society

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3.
Meeting: Third International Congress of Bronchoesophagology.
Place: Lisbon, Portugal. Time: September or October, 1954.

International Congress of Audiology

Place: Leyden, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

International Course in Paedo-Audiology

Place: Groningen, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

Pan American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

Meeting: Fourth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology
Place: México, D. F., Mexico. Time: Feb. 28-March 4, 1954.
President: Dr. Justo Alonso.
Exec. Secretary: Dr. Chevalier Jackson, 1901 Walnut St., Philadelphia 3.
President of Congress: Dr. Ricardo Tapia Acuna.

Third Latin-American Congress of Otorhinolaryngology and Bronchoesophagology

Place: Caracas, Venezuela. Time: Feb. 21-25, 1954.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Dean Lierle, Iowa City.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Place: San Francisco.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. James Milton Robb, 315 Lakeland Ave., Grosse Pointe 30, Mich.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago.

AMERICAN BOARD OF OTOLARYNGOLOGY

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Statler Hotel, Boston. Time: May 10-15, 1954.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Secretary: Dr. F. Johnson Putney, 255 S. 17th St., Philadelphia 3.
Place: Statler Hotel, Boston. Time: May 25-26, 1954 (afternoons only).

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Statler Hotel, Boston. Time: May 20-21, 1954.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
President-Elect: Dr. Kenneth M. Day, 121 University Place, Pittsburgh.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Statler Hotel, Boston. Time: May 25-27, 1954.

SECTIONS:

Eastern.—Chairman: Dr. Daniel S. Cunning, 118 E. 53rd. St., N. Y. 22.
Place: Hotel Syracuse, Syracuse, N. Y.
Southern.—Chairman: Dr. William C. Wolfe, 332 W. Broadway, Louisville 2.
Place: Andrew Jackson Hotel, Nashville, Tenn.
Middle.—Chairman: Dr. French K. Hansel, 634 N. Grand Blvd., St. Louis 3.
Place: The Drake, Chicago.
Western.—Chairman: Dr. Guy L. Boyden, 1735 N. Wheeler Ave., Portland 12, Ore.
Place: The Elks Club, Los Angeles.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: Statler Hotel, Boston. Time: May 16-17, 1954.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

President: Dr. Harry Neivert, 555 Park Ave., New York 21.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

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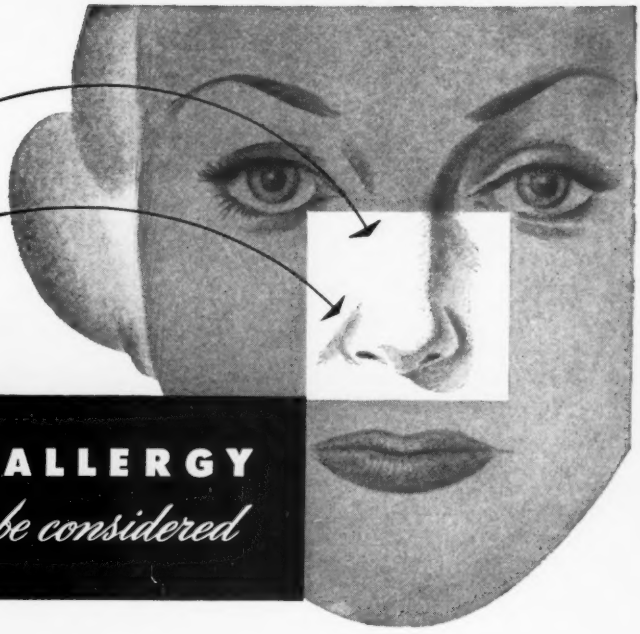

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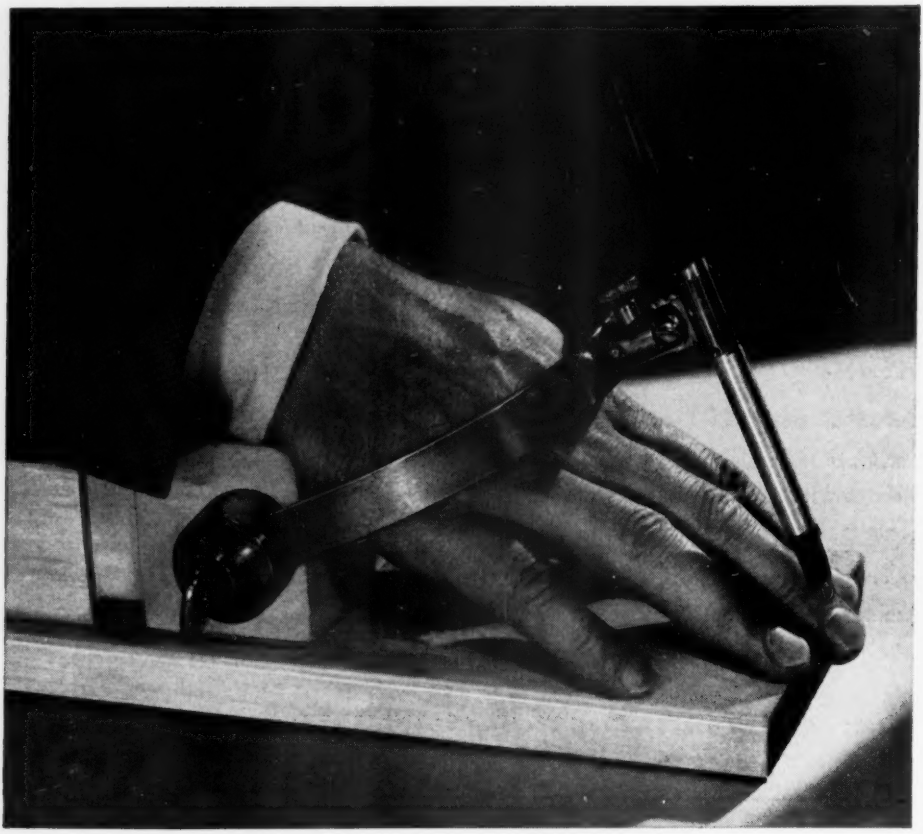

with other cigarette filters

To compare the efficiency of various filters as they affect physiological responses in the cigarette smoker, drop in surface skin temperature at the last phalanx was measured.

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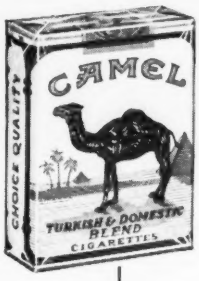

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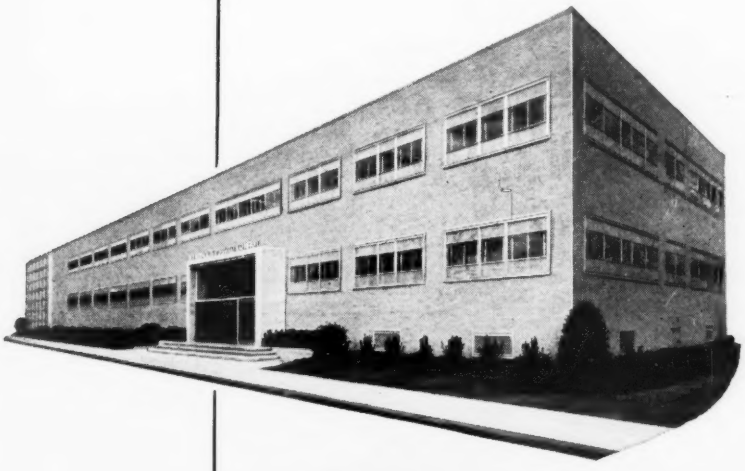

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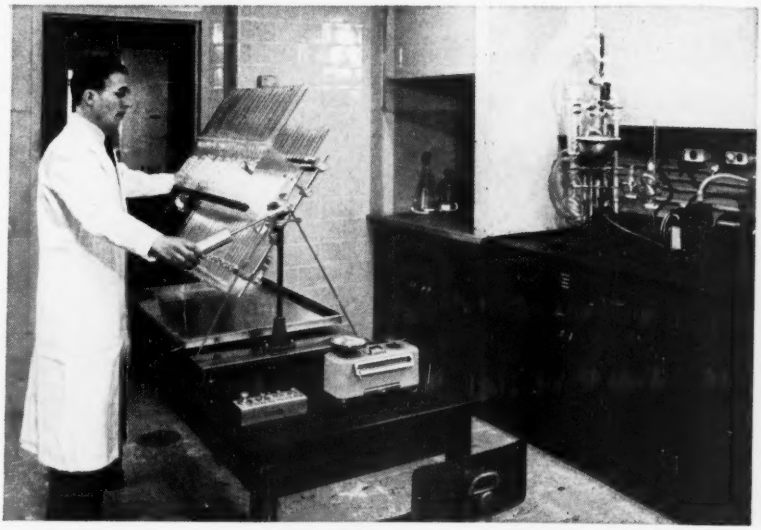

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